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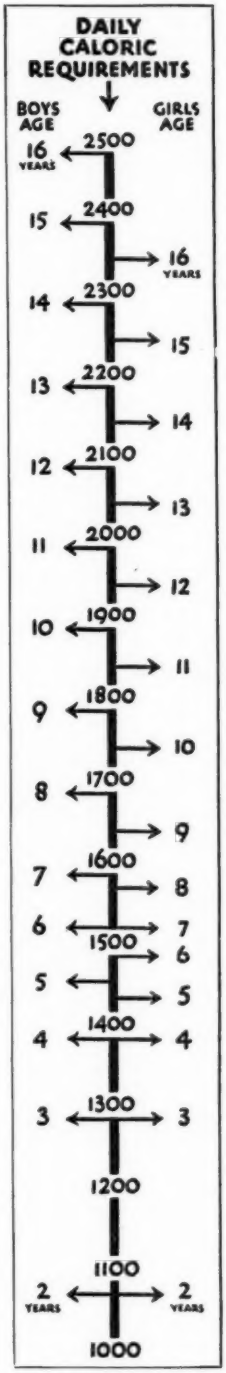

Figures from Kugelmass's "Feeding in Infancy and Childhood"

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Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society.

Volume 18 November, 1935 Number 11

THE PREVENTION OF WHOOPING COUGH WITH BACILLUS PERTUSSIS VACCINE*

LOUIS SAUER, M.D.

Associate in Pediatrics, Northwestern University Medical School

Chicago, Illinois

WHOOPING cough: A specific, endemic infection of the respiratory tract, most prevalent in young children, is characterized by persistent paroxysms of coughing which, in the course of weeks, usually end in an inspiratory whoop and vomiting; the resulting immunity is usually permanent.

The pertussis bacillus, isolated, cultivated and accurately described, in 1906, by Bordet and Gengou[1] of the Brussels Pasteur Institute, fulfills as the etiologic factor the postulates of Koch. These investigators found this minute bacillus, at times, in nearly a pure state, in the glistening pearls of mucus, coughed up early in the disease. This Gram-negative, capsulated, hemoglobinophile is notoriously difficult to isolate. It grows well on the culture medium perfected by Bordet.† A well-developed colony approximates one millimeter in diameter. When viewed in reflected light, a definitely darkened zone is usually seen to surround each minute, round, markedly raised, pearly gray colony. In transmitted light this darkened area, and the colony, appear distinctly lighter than the surrounding medium. The soft colony can be quite completely wiped up with one sweep of the platinum loop. In subculture, the growth is usually rather luxuriant within forty-eight hours. The organisms diffuse evenly when mixed with a droplet of water on a clean slide. Microscopic smears stain feebly. When first isolated, some of these ovoid bacilli show bipolar staining. In frequent transfers they usually appear more coccoid. Unlike B. influenzæ, pleomorphism is rare in recently isolated cultures.

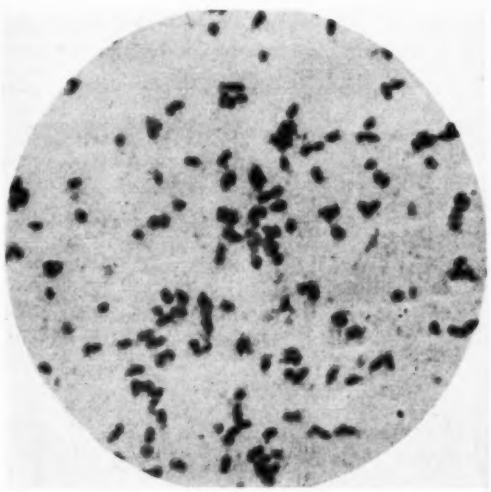

Fig. 1. Bacillus pertussis × 1740.

Inaba,[4] in 1912, reported the first authentic case of experimental whooping cough. Two weeks after a freshly isolated, forty-eight-hour growth of B. pertussis was swabbed on the pharynx, the ten months' old macacus monkey began to cough in paroxysms. On the seventeenth day, severe paroxysms of coughing ended with the expectoration of mucus and vomiting. A Gram-negative bacillus was recovered. The

*Read at the 82nd Annual Meeting of the Minnesota State Medical Association and before the Medical Section, American Association for the Advancement of Science; Minneapolis, June 25, 1935. Presentation sponsored by the Northwestern Pediatric Society.

†Boil until soft 500 grams of peeled, sliced potatoes in 1,000 c.c. distilled water and 40 c.c. glycerin. Adjust to original weight and strain through gauze. To 500 c.c. of the filtrate add 1,500 c.c. of 0.6 per cent salt solution and 60 grams of agar. After autoclaving, cool to 45° C.; add 120 c.c. of the supernatant to 30 c.c. of sterile, fresh defibrinated blood (human, sheep or horse) warmed to 45° C. Pour into sterilized Petri dishes, culture boxes or tubes; refrigerate until used.

complement fixation test was positive, when the cough subsided. After many futile attempts, Sauer and Hambrecht[8] produced paroxysmal coughs from one to three weeks after recently

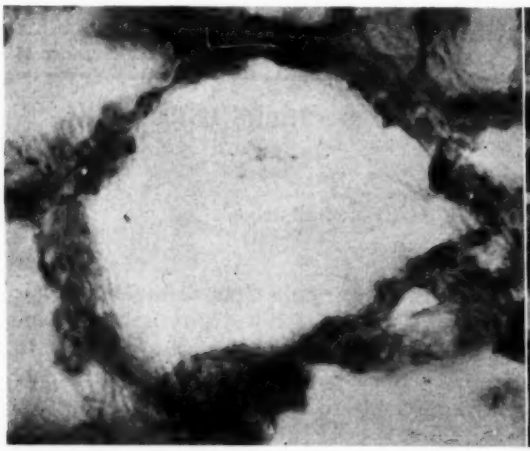

Fig. 2. Alveolus in benign experimental pertussis. Alveolar walls show no intracellular inclusion bodies. × 1500.

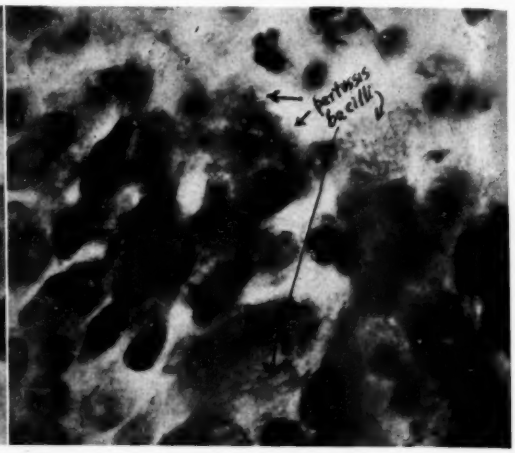

Fig. 3. Human pertussis bronchopneumonia. Alveolus contains: B. pertussis, lymphocytes, leukocytes and desquamated epithelial cells; no intracellular inclusion bodies were found. × 1500.

isolated B. pertussis cultures were injected into the larynx of five healthy young monkeys. Similar coughs followed inoculation into the nares of three healthy young monkeys. The white cell and differential blood counts usually showed the transient lymphocytosis characteristic of pertussis in man. The pertussis bacillus was recovered from deep throat cultures; also from the larynx, trachea and lungs, after death. During the height of the cough, the bacillus was recovered from an animal that had been inoculated with the bacillus recovered from the lung of another coughing animal. Animals that recovered were immune to subsequent overwhelming injections of various strains of B. pertussis. The bronchi were found to be the primary seat of the infection in uncomplicated (benign), experimental pertussis.[9] Endobronchitis and peribronchitis were more pronounced than endobronchiolitis and peribronchiolitis. The latter were more pronounced in human and in experimental pertussis bronchopneumonia. Alveolar involvement seemed to occur by way of the bronchioles. Only pertussis bacilli were found on and between the cilia of the finer bronchi in benign experimental pertussis. In human and in experimental pertussis bronchopneumonia, pertussis bacilli were found also in the alveoli. Mucopurulent inflammation was replaced relatively early by round cell infiltration; the latter was most pronounced where the injury was most profound. Typical intracellular inclusion bodies could not be found.*

The diagnosis of whooping cough is relatively simple when the cough is already typical, or when it is known that a coughing non-immune was intimately exposed to infection. If the paroxysmal cough has persisted for more than a week, white cell and differential blood counts often aid in diagnosis. Leukocytosis over 15,000, with an increase in the percentage of lymphocytes, is characteristic of pertussis; but the absence of such changes in the blood picture does not exclude whooping cough. The cough plate of Chievitz and Meyer,[2] of the Danish State Serum Institute, when positive, is a valuable laboratory aid in early diagnosis. Negative plates, however, do not exclude the disease. If a Petri dish or culture box of fresh Bordet medium is held, vertically, in front of the open mouth at the moment of several deep, expulsive coughs, and promptly incubated at 37° C., the minute colonies of B. pertussis can often be distinguished with a hand lens at the end of three

*Recent experimental work on monkeys by: Shibley, G. S. (Proc. Soc. Exper. Biol. & Med. 31:576, 1934); Rich, A. R., P. H. Long, J. H. Brown, E. H. Bliss, and L. E. Holt (Science 76:330, 1932); Culotta, C. S., D. F. Harvey and E. Gordon (J. Ped. 6:743, 1935); seems to rule out filtrable virus and substantiates the etiological role of B. pertussis.

or four days. Only properly exposed plates should be incubated. Satisfactory exposure, sufficiently early in the disease, is the most important technical step. At the daily examination of the incubated plate, rapidly growing saprophytes should be removed with the hot wire. In a total of 4,432 cases of pertussis, cough plates were exposed by seven groups of workers, with the following percentage of positive plates: first week, 77; second week, 61; third week, 56; fourth week, 30; fifth week, 14; sixth week, 3.

Specific serologic tests, such as agglutination and complement fixation, usually become positive some weeks after the onset of symptoms, reach a peak, then decrease or completely disappear within a few months or years. Their course is quite similar after the injection of non-immunes with potent antigen. Huenekens[3] was the first to show that fresh B. pertussis vaccine produces a positive complement fixation test. Such tests, however, are no index of immunity. They are transient, whereas immunity is more prolonged—usually lasts for the remainder of the individual's life. Indeed, when such tests are at their height, active immunization is not yet complete. A time interval of several months must elapse before the injection of 8 c.c. of this B. pertussis vaccine will produce active immunity in a majority of exposed young non-immunes.

Nearly a score of investigators have reported specific immunity tests (*i.e.*, a skin-test like the Schick test), whereby non-immunes can be differentiated from immunes, but confirmatory evidence is still lacking. The whooping cough bacillus is an endotoxin-producing pathogen which imparts relatively little soluble toxin to the culture medium in which it grows.

Although exposure to infection is not synonymous with infection, it serves as the ultimate test of the efficacy of an immunizing agent. The evidence of active immunization with B. pertussis vaccine is based on what happens to injected nonimmunes when subsequently exposed to whooping cough. Because exposures in the family are usually more intimate, repeated and prolonged, than are casual exposures outside (as in the classroom or at play), exposures are grouped into: "familial" and "outside." One cubic centimeter of the vaccine is injected, hypodermically, in the deltoid region of each arm; a week later, 1.5 c.c. are injected into the biceps region of each arm; a week thereafter, 1.5 c.c. are injected into the triceps region of each arm. Syringe and needles are sterilized by heat (oven

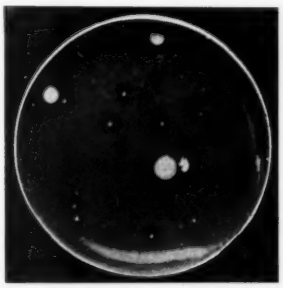

Fig. 4. Positive cough plate showing zoned colonies.

at 250° F. for one hour). After briskly shaking the vial, the rubber cap and site of injection are sterilized with sterile cotton saturated with ethyl alcohol. Parents should be forewarned of the transient local tenderness, redness and occasional rise in temperature; also, of a local nodule or induration which may persist for several weeks. Injections should not be given in the area of a previous toxoid injection or smallpox vaccination. Parents should not take the child's temperature, nor should anything be applied locally. Over 10,000 individual injections of this vaccine have been given, in Evanston, without producing an abscess or scar. Should a marked reaction occur, the subsequent doses should not be increased, but an extra bilateral injection should be given a week after the last injection, so that the total dosage equals 8 c.c. (1 c.c. in each arm for four successive weeks).

Between 1928-32, a total of 394 selected young non-immunes (average age about fourteen months) were injected with the vaccine made at the Evanston Hospital.[7] To date, a total of 30 of these injected children in twenty-four families were intimately exposed to whooping cough in their twenty-eight non-injected (control) brothers and sisters. All of the controls in these families contracted the disease; *i.e.*, the communicability rate was 100 per cent. All of the injected children escaped. A total of 167 of the injected children were casually exposed over 200 times, outside the household, but none contracted the disease.

Northwestern University Medical School has granted to two biological laboratories the privilege to make the vaccine according to our detailed specifications. Each month they are supplied with recently isolated, hemolytic strains. Human blood is used in the culture medium. Since 1932, over 1,000 young non-immunes (average age about eleven months) were injected with the approved commercial vaccine. This group is comprised of about 450 private patients, 500 city welfare infants, and 160 in a Chicago orphanage. There have occurred, to date, thirty-eight familial exposures, with three failures; also, thirty-one casual (outside) exposures, with three failures.

Since 1932, twenty-nine physicians in eighteen states have reported the injection of 2,348 young non-immunes. The average age of this group is appreciably older. A total of seventy-six subsequent exposures are known to have occurred. To date, thirteen of the injected children have contracted the disease. It is not unlikely that the number of exposures was appreciably higher than seventy-six. The percentage of failure, computed from the number of known exposures, is 17.1 per cent. From a total of approximately 3,800, a total of 342 are known to have been exposed with a total of nineteen failures; *i.e.*, a protection of over 90 per cent. Most of the injected children who contracted the disease had it in a mild form.

The most conclusive evidence that the Evanston vaccine produces active immunization was reported by Macdonald and Macdonald,[5] in 1933. They injected two of their four non-immune sons with the Evanston vaccine. Five months thereafter, when the four boys had been in quarantine for several weeks (to exclude outside infection), a very weak suspension of living pertussis bacilli was inoculated, intranasally, into each of the four boys. The non-injected boys contracted typical pertussis (positive cough plates, lymphocytosis, paroxysmal cough, whoop and vomiting for weeks). The injected boys escaped; neither developed a cough from the inoculation of living pertussis bacilli, nor from the intimate exposure to their coughing brothers. At the conclusion of the experiment the complement fixation was equally positive (four plus) in the four boys. This test brings strong evidence that whooping cough is due to the Bordet-Gengou bacillus; also, that the Evanston vaccine produces active immunity.

Because failure of the vaccine to confer active immunity may be due to a number of causes, it seems advisable to discuss this in greater detail.

1. Mishulow, Oldenbusch and Scholl[6] found room-temperature to decrease the antigen in B. pertussis vaccine. Since warmth accelerates chemical changes, deterioration is retarded by keeping the vaccine cold (at 8 to 12° C.) until it is used.

2. One of our failures was in a girl of five years. The other three children of the family, immunized at the same time, escaped, although very intimately exposed to her throughout her illness. It is not unlikely that children over three or four years of age may require somewhat more than 8 c.c., possibly 10 or 12 c.c., of the approved commercial vaccine. Several physicians, who kindly reported failures, offered this suggestion.

3. Three of our failures occurred in children who were injected soon after recovery from measles. Until more is known about the effect of such disease on the immunity response from pertussis vaccine, it seems advisable to wait a month (or more) after recovery, before giving the vaccine. The occurrence of other diseases soon after giving the vaccine might likewise inhibit or deter the immunizing effect of the vaccine.

4. It has been quite definitely established that a time interval of several months is required for active immunity to be completed. If exposure to infection occurs within that time, failure might occur. Several parents have wilfully, but without our knowledge, intimately exposed their five injected children to early cases of whooping cough, more than a year after the injections, without producing the disease.

5. The potency of the individual strains and the strength of the vaccine (1 c.c. should approximate 10,000 million bacilli) play important rôles in the immunity response.

Because whooping cough is more likely to be serious during the first three years of life, it seems rational to give the pertussis vaccine first, *i.e.*, before the other customary immunizations. Until it has been determined how early in life active immunization can be effected, the best age seems to be between the sixth and twelfth months —preferably during the eighth month. The following plan has been found practical: to vaccinate against pertussis at eight months; against

diphtheria at a year; against smallpox at eighteen months (when the subsequent Schick test readings are made); and against scarlet fever before nursery-school or kindergarten attendance. Because several months are necessary for the immunity response to be completed, other immunizations have not been given within four months after the pertussis vaccine injections; nor has the vaccine been given until several months after other immunizations. Results will depend upon: the concentration and quality of the vaccine; the vigilance of physician, jobber and druggist in regard to the storage and age of the vaccine; the age and health of the child when the vaccine is given; and the time interval between injection and exposure.

Conclusion

This bacillus pertussis vaccine should prevent whooping cough in a high percentage of young non-immunes, if the outlined details are observed.

References

1. Bordet, J., and Gengou, O.: Le microbe de la coqueluche. Ann. de l'Inst. Pasteur, 20:731, 1906.
2. Chievitz, L., and Mayer, A. H.: Further investigations on whooping cough. Ann. de l'Inst. Pasteur, 30:503, 1916.
3. Huenekens, E.: The prophylactic use of pertussis vaccine controlled by the complement fixation test. Am. Jour. Dis. Child., 14:283, 1917.
4. Inaba, I.: Ueber den Bordet-Gengouschen Keuchhustenbacillus bes. Uebertragungsversuche des Keuchhustens auf Treve. Ztschr. f. Kinderh. 4:252, 1912.
5. Macdonald, H., and Macdonald, E.: Experimental pertussis. Jour. Infect. Dis., 53:328, 1933.
6. Mishulow, L., Oldenbusch, C., and Scholl, M.: Potency of stored pertussis vaccine. Jour. Infect. Dis., 41:169, 1927.
7. Sauer, L.: Immunization with Bacillus pertussis vaccine. Jour. Am. Med. Assn., 100:239, 1933; 101:1449, 1933.
8. Sauer, L., and Hambrecht, L.: Experimental site of lesion. Arch. Path., 8:944, 1929.
9. Sauer, L., and Hambrecht, L.: Whooping cough; site of lesion. Arch. Path., 8:944, 1929.

THE INCEPTION AND DEVELOPMENT OF FLUOROSCOPY: THE INFLUENCE OF CARMAN ON ITS STATUS IN AMERICA*

PERCY BROWN, M.D., F.A.C.P., F.A.C.R.

Boston, Massachusetts

MERELY to have been considered without disfavor in connection with the second Russell Carman memorial lecture is compliment indeed; to have been chosen to deliver it is distinction of the first order. But as I stand before you thus distinguished, I feel impelled to declare that the essential source of my appreciation lies even deeper—most profoundly do I esteem also the privilege of taking part in this hour so appropriately commemorative of the forthright life and good works of a former colleague and an old friend. His consistent progress along the clearly-cut path of his professional life from the moment of our first meeting I observed with pride—pride in the possession of his friendship, and pride on behalf of my specialty of radiology in *its* possession, in him, of a champion of its most exalted purposes and ideals. In view of the years of his distinguished identity with the activities of his life-work, culminating with his world-recognized eminence in it, strange and fantastic seems the thought of his once serious interest in any other denomination of medical endeavor, yet in this connection the circumstances of our first meeting will ever be before me in memory, and they may be of a moment's passing interest.

I first saw Russell Carman seated on a low bench in a darkish corner of the basement orthopedic plaster-room of one of Boston's smaller hospitals. His legs were extended and his chin was sunk upon his chest. But his posture implied neither dejection nor profound meditation—far from it. He was merely carving the lines of a proper longitudinal arch on the plaster cast of a flat-foot. At me, to him a stranger, he looked up with that smile that no visitor to Rochester in later years will ever forget, tapping the cast with his knife-handle to remove the shavings; and with his spoken pleasantry to the effect that

*The Russell Carman Memorial Lecture for 1935, presented at the annual meeting of the Minnesota State Medical Association, Minneapolis, June 25, 1935.

a second Praxiteles had arrived to grace the world of sculpture, our friendship was born—vivified, no doubt, by being swathed in the warming folds of his spontaneous humor. *Apropos* of Carman's occupation at that moment, Doctor Balfour told you last year of his original intention to enter upon the practice of orthopedics. He would have adorned that branch of surgery even as he graced the field that ultimately he chose, but, in the light of historical record, what a loss to the development of radiology had Carman adhered to his first intention! The Fates, however, in a moment of kindly disposition placed him, rather, in a field of Medicine the soil of which had been not long turned over by the ploughshare of discovery, and which was awaiting the energies of those who would seminate it and would cultivate it in a manner best suited to the welfare of Humanity. Russell Carman, by fortune's favor and his own integrity of purpose, became one of these, and that which he accomplished is inscribed on many a fair page of the history of scientific progress, which need no re-reading to this audience.

Carman was ever a protagonist for fluoroscopy in roentgen diagnosis both by word and deed. Throughout the major portion of his medical life, his published observations were based in generous measure on his findings by fluoroscopic screen, notably with reference to alimentary anomaly and disease. As he preached so did he practise. A fluoroscopic demonstration by him was one of the Rochesterian lodestones that have for so long projected their magnetic attraction into all corners of a medical world that delights to learn by observation. To sit by his side, facing the pale glow of his fluoroscopic screen, was to witness the exhibition of a skill that could result only from his deep love of the work as well as from his implicit faith in it. In Carman's hands, the practice of fluoroscopy involved the exercise of a sympathetic individualism that would be quite lost amid the multi-personal technicalities of mere radiography. What better proof of this than his memorable habit of preliminary kindly inquiry as to the habitation, home-life and little predilections of an agitated patient who had entered for the first time the Cimmerian atmosphere of the screening-room, but who, from that moment, was disturbed no longer and whose upper digestive tract forthwith shared his tranquillity? It is an *art* thus to mitigate, with the warmth of sympathy, the cold impersonality of an applied science—and in the doubtful gloom of that chamber, in front of an invalid soothed and calmed without having even seen clearly his benignant face, sat an artist of the first magnitude!

Perhaps, then, it were not inappropriate, as the second lecture to be given in respectful commemoration of the life of this remarkable man, briefly to consider the history of the diagnostic adjuvant by which he laid such great store, which he was ever ready stoutly to defend, the elaboration and expansion of which his loyal support so greatly aided, and through which he served humanity so devotedly and so effectively.

* * *

It is to the gifted Irish Cantabrigian, Sir George Gabriel Stokes, of course, that we are indebted for the term *fluorescence* as well as for the greater part of our present knowledge of this subject, but we should not allow to escape from our memory the name of the discoverer of this beautiful phenomenon as well as of the principle of lenticular stereoscopy and of the kaleidoscope, the versatile Sir David Brewster, Scottish theologian, natural philosopher, biographer and editor. Well-known are his observations on the behavior of a beam of solar light on passing, after condensation, through a solution of chlorophyll. The sub-fluid course of this beam was revealed to him as a ray of brilliant crimson. Investigating further, he observed varied color-change, produced in the same manner, in connection with other vegetable solutions, with certain essential oils and also with some crystals, among them native calcic fluoride, or fluor-spar. The results of this initial experimentation were communicated by him to the Royal Society of Edinburgh in 1833. Calling the phenomenon *internal dispersion*, Brewster believed it due to the scattering of light by colored particles held in suspension. Somewhat later, Sir John Herschel observed that a beam of solar light, striking a solution of quinin sulphate, became "epipolized," as he called it; it assumed a blue color in a stratum just beneath the surface of the fluid at which the beam entered. Beneath this stratum the beam was not changed as to color or strength, nor could it be recolored by passing it through a second solution of quinin sulphate.

As much later as 1852, it remained for Stokes,

using at first a solution of quinin sulphate, to show that the effect was due, not to the scattering of light by suspended particles, but to the fact that the dispersed beam actually differed in refrangibility from the light which excited it. On this basis, he called the phenomenon for a time *true internal dispersion,* as opposed to the theory of light scattering, which he considered under the name of *false internal dispersion.* Ultimately dissatisfied with this definition, he adopted the term *fluorescence.* The outcome of Stokes's prolonged experimentation is now common knowledge of the physical laboratory; it resulted in the law that the fluorescent light is of lower refrangibility, of greater wave-length, than the light exciting it, or, as the electrophysicist Hammer[10] many years later aptly observed: "In electrical parlance, a fluorescent substance might be termed a step-down transformer for light-waves." Stokes' Law did not apply to all substances, as he believed, for it was later shown that there are more than one kind of fluorescence. Of the scores of substances from the mineral and vegetable kingdoms that had contributed to the researches of Brewster, Herschel, Stokes, Pierre, Lommel, Voight and others, the humble group of the platino-cyanids alone were touched by the bright wand of Destiny, and down through the long years of laboratory experimentation and lecture-room demonstration the platino-cyanid of barium found its solitary way to that memorable table in one of the smaller rooms of the Physical Institute of the University of Würzburg. Heretofore, the phenomenon of fluorescence was an instrument of pure science, largeiy utilized to illustrate the laws of reflection and refraction. The instant a bit of paper coated with this salt was seen to glow in complete darkness, for no apparent reason at the moment, by the quick eye of Professor Röntgen, rays of a wave-length otherwise invisible to the eye were revealed, and at the later moment when Röntgen first interposed an object between this fluorescent paper and the Hittorf-Crookes tube, encased in black paper, with which he was just then working, fluoroscopy as a practically living thing was born.

Röntgen, according to the story that has been retold so vividly of late,[9] had been engaged in repeating Lenard's experiments with cathode rays, and had covered a Lenard tube with black paper to observe their fluorescence through a window in the tube. He then continued his work with a Hittorf-Crookes discharge tube. The barium platino-cyanid paper was at hand because, as he later told Mackenzie-Davidson, this salt was in common use among German physicists to reveal the invisible portions of the spectrum. Röntgen's use of it in his succeeding observations, once its behavior in the presence of the covered tube was noted, was mentioned by him in his classic *Preliminary Communication.* This resulted in its almost universal adoption as the medium of choice in the earliest days of fluoroscopic investigation. In its employment the method of Röntgen was generally followed, except that there was a widespread effort to obviate the darkened chamber enclosing the observer. Curiously enough, in their enthusiasm over their specialized devices to simplify the scrutiny of the fluorescent screen by merely shading the eyes, many physicists almost created a popular conviction that they had produced something involving an entirely new application of the principle of x-radiation. This sentiment was perhaps strengthened by the fact that each was quick to give his device a specific name. Thus, as the painstaking historian Glasser observes, the "cryptoscope" of the Italian physicist Salvioni,[13] of Perugia, "caused almost as much enthusiasm . . . as was caused by the announcement of the discovery of the roentgen rays." Yet it would be unjust to the spirit of Salvioni to assume that he sought for himself one jot or whit of such credit, unthinkingly misplaced. His instrument, the first of its kind, was tubular in form. One end was closed by a sheet of black paper upon which, for temporary lack of barium platino-cyanid, was spread a layer of fish-glue and calcium sulphid. Within the tube, near the opposite end, was fixed a lens giving a clear image of the luminescent paper, which must have been so largely phosphorescent. Concerning this device, Salvioni was modesty itself. "In this," he says in his description, "there is, of course, nothing new which could not have been deduced from the original experiments of Röntgen; the novelty, if indeed, it is so, consists merely in making use of the known facts to design the arrangement. It seems to me that, in a more perfected form, it might be of extensive use in surgical and medical science. The sulphide of calcium may be replaced with ad-

vantage by the cyanide of barium and platinum . . . "

Salvioni's term "cryptoscope" was not infrequently applied to any form of hooded screen for some years following 1896. One well remembers its occasional usage in our country even after the period of Edison's exhaustive investigations of that year. William Rollins very frequently made use of the term. Close on the heels of Salvioni's work came reports of other forms of hooded fluoroscopic screens. The instruments of Spies and E. P. Thompson were truncated cones instead of being cylindrical. Thompson, not to be outdone in the way of especial designation, called his device a "kinetoskotoscope." The apparatus of Magie, of Princeton University, was closely contemporaneous with that of Salvioni; likewise it was tubular in form. It was known as a "sciascope." The devices of John MacIntyre and of Edison were truncated pyramids; it was this form that prevailed in the later construction of the hooded fluoroscopic screen, through the romantic and tragical days of its rise, decline and fall.

Throughout what may be called the pre-Edisonian era, all designs of fluoroscopic apparatus involved the use of the platino-cyanid of barium. Salvioni mentions its desirability and undoubtedly would have employed it in his first device had circumstances permitted. An early exception was the screen of Herbert Jackson, of Kings College, London, upon which potassium platino-cyanid was the salt employed.

From Mr. Edison's well-known experiments of 1896 with a tremendous number of crystalline substances came the discovery of a fluorescent salt which in many ways, although not in all, was superior to the cyanid; it was not so susceptible to atmospheric conditions but it was sufficiently phosphorescent to give it an appreciable "lag," but for fluoroscopic purposes this was not a great fault. With this substance, calcic tungstate, in a finely divided form, he coated a plane support and covered this screen with a hood in the shape of a truncated pyramid to exclude daylight, placing an aperture formed for the eyes at the line of truncation and a supporting handle on one of the sides of the pyramid. Edison called his device a "fluoroscope," but in declaring that he invented the fluoroscope many historical writers have created the false impression that he was the discoverer of the initial practical means by which invisible x-rays are changed to visible light rays. This discovery, of course, was Röntgen's, but to Edison is to be given the credit of the discovery of the tungstate of calcium as a fluorescent material of peculiar qualities of excellence.

The use of the hooded screen had as its earliest purpose the lecture-room or laboratory identification of the presence of roentgen rays in the general study of radiation; later, it was frequently used in demonstrations that were held under conditions of a character less academic, many of which were patterned, on a smaller scale, after Edison's historic fluoroscopic exhibition of 1896, given in conjunction with the exposition of the National Electric Light Association at the Grand Central Palace, New York. Also, the hooded screen was used as a medium of gauge to test the quality of x-emanation during the course of evacuation of Crookes tubes or of radiographic exposures—a pernicious practice to which is appended one of the sad phases of radiologic history. Finally, as the petals of the flower of evolution slowly opened, the *fluorescent* screen of the physicist became the *fluoroscopic* screen of the clinician, in form both open and hooded, and the path was cleared for its steady advance in diagnostic usefulness.

There can be no question that the earliest medical attitude toward fluoroscopy, or roentgenoscopy, was in great measure influenced by the contemporaneous opinion of the physical or engineering laboratory. In view of the practical outcome of his intensive labor in the matter of fluorescent substances, Mr. Edison was enthusiastic, even arbitrarily so, over the future of his "fluoroscope." Said he, as reported[8] in the *American Journal of Photography* of April, 1896, ". . . There is no occasion to take photographs, shadowgraphs or radiographs. I stopped that long ago. You can see for yourself, the fluoroscope does the work in a moment." One can not but wonder at the effect on the development of radiology had he indeed "stopped" roentgenography! No doubt he had in mind, in this connection, the problems of minor traumatic surgery which, at an earlier day, usually presented themselves to non-medical thought where the roentgen rays were concerned, but Edison's every pronouncement attracted wide attention and one is constrained to feel that his attitude had much to do with the intensity of the "first fluoroscopic

era," as it may be called, that obtained from 1896 to 1900. There is but little doubt, also, that the demand upon the early roentgen worker of this period for the "quick" use of the fluoroscope (which, mind you, was still largely the hooded screen) in emergency surgery or in the more gross pathology of the thorax, was at least one of the factors, albeit a lesser one, making for the wide incidence of radiation-dermatitis during those years. "All we need here is the fluoroscope—that will tell us all we want," are words that still ring in the ear of the medical pioneer in radiology, and, since the use of this instrument alone was thus preordained, the ensuing scrutiny was usually prolonged. In this way the seed of irreparable damage was frequently sown. The martyred pioneer Kassabian, in somewhat rueful retrospection of the conditions of the first fluoroscopic era that led to his plight, remarked of the ardent souls who thus importuned him: "I would have stopped the [use of the] fluoroscope then, but they would not let me." However, the bearing of the fluoroscope on the history of the martyrs to roentgenology is a matter of another writing, and by no means germane to our consideration of today.

Francis Williams of Boston, constantly aided by his *fidus Achates*, William Rollins, labored diligently throughout these four intensive "fluoroscopic" years. Being an internist by medical training and for many years an attending physician to his hospital, he paid little attention, when once launched upon his new-found work with x-rays, to the clamor for the impromptu use of the fluoroscope in minor surgical problems. Williams and Rollins, by nature, were to hasty and incomplete procedure as oil, by nature, is to water. This was the existing equation, at the Boston City Hospital, with reference to the first importunate demands for immediate resort to the fluoroscope in almost any clinical situation. But the rational applications of the fluoroscopic screen attracted Williams at once, and some months later he wrote: ". . . it was the confidence I had in the possibilities of the fluoroscope to assist in the diagnosis of diseases of the lungs and heart that led me to begin and develop this method of examination . . ."[15] It was this internist's interest in the thorax and its topographical opportunities that resulted in his employment of the *open* screen; perhaps the first consistent use of this implement in this country. His predilection for the open screen was due also to his practice of making tracings, on glass, celluloid or the patient's skin, of his observations covering the entire thorax. For this at least one free hand was needed, and often two, for we must remember that foot-control of current-supply was unknown at that day, diaphragm-control was not to be exercised easily or quickly, and the behavior of the tube was governed by manipulation of bi-polar series spark-gaps controlled by rods. The infant focus-tube was usually obstreperous, and to "spare the rod" was to "spoil the child."

Williams' devotion to the fluoroscopic use of roentgen-rays naturally broadened his viewpoint as an internist to a degree quite beyond his time, because he perceived how the diagnostic powers of even the most skilful clinician might be amplified and refined thereby. "It has been said," he quoted in 1901 with thorough acquiescence, "that the 'x-rays are a most effective method of showing how great a rôle the imagination may play when using auscultation and percussion.'" By 1898, hardly over two years from Röntgen's discovery and through the medium of the fluoroscopic screen, Williams had discussed the application of the x-rays to the diagnosis of thoracic aneurysm, pericardial effusion, cardiac hypertrophy, cardiac transposition, emphysema, pleurisy with large or slight effusion, pneumothorax, hydropneumothorax and pulmonary tuberculosis.

Williams, of course, shared the common experience of the pioneer, whatever the undertaking. His belief and confidence in the revelations of the fluoroscope courted skepticism and even antagonism among his clinical colleagues. One of his publications on the subject was rewarded by this editorial appendage to a review of it:[16] "The author is evidently an enthusiast. It is unfortunate that he has confined himself so largely to work with the fluoroscope. Observations made in this manner are liable to errors of personal interpretation . . ." As if, forsooth, the findings derived from other clinical methods, not excepting radiographic methods, were not, on frequent occasion, disposed to the same weakness!

One cannot contemplate the development of roentgenoscopy without recurring to the contributions of that ingenious dental practitioner, William Rollins, whose name should have adorned the honorary roll of every radiologic society in

the land. How many of us today are aware that he devised and used practically an intra-oral fluoroscope for the teeth, manipulated in the manner of the mouth-mirror? But it was as the faithful co-worker and mechanical genius of Francis Williams that he produced the extraordinary instrument he called the "seehear"—a fluorescent screen and a phonendoscopic diaphragm of equal size mounted in such relation to each other that the eye and ear of the examiner could appreciate conjointly the sights and sounds of the intrathoracic functions, or pathological departures from them.

Crane, of Kalamazoo, was another advocate of fluoroscopy in thoracic diagnosis whose contemporaneous enthusiasm was almost the equal of that of Williams. This was shown in a communication made by him as early as 1899,[7] in the course of which he describes his "skiameter," which, as its name implies, was a manual instrument for the mensuration of shadow-densities by means of a gauging scale, at first of metallic wires of varying cross-section and later of layers of tin-foil arranged in divers thicknesses. The depth of the given shadow beneath the screen surface could in this manner be determined from its intensity by an ingenious, if somewhat complex, application of mathematics. Crane's confidence in fluoroscopy was revealed by this sentence: "I trust that in this paper may be shown the superiority of the fluoroscope over the dry plate [the radiograph] in the examination of the lungs."

Williams and Crane, of course, were but two of a widely disseminated body of investigators that was moulding the advantages of scrutinizing inspection to the needs of thoracic diagnosis by means of the roentgenoscope, as it was now to be called, for, since the advent of the open screen, the term "fluoroscope," in the Edisonian sense, was no longer a comprehensive one. In this connection we should recall the names of Grunmach, Kienbock, Walsham, Holzknecht, Stubbert, Moritz, Levy-Dorn and Groedel, as well as that of Baetjer and certain of his American colleagues of a slightly later day. It is safe to say that some of these, by their support of thoracic fluoroscopy in terms of reason and conservatism, retarded the decline of the use of the roentgenoscope, while others, of more ardent enthusiasm, accelerated it—for a decline there was during the very early years of the present century. This reaction was the result of at least two influences, one of which was the increasing realization, especially among practical users of roentgen rays, that the fluorescent screen could be the source of very real danger to the operator. The first death from the effect of x-radiation in the United States, that of young Clarence Dally, an artisan of Mr. Edison at his West Orange laboratory, was recorded in 1904, and as late as 1905, Pfahler,[12] in a paper on the roentgen diagnosis of diseases of the lungs, admits his practical discontinuance of the use of the fluoroscope in the chest "because of its inaccuracy and of the dangers attending its use." Another influence making for such sentiment adverse to the practice of fluoroscopy was the steady improvement in radiographic methods, as shown by the creation of generating apparatus of more potent output and the development of intensifying screens of better quality; that is to say, of less granularity and phosphorescence.

But Research, in the meantime, remained true to her renown for courageous and persistent endeavor in the face of obstacles. Her devotees, so definitely aided by fluoroscopic methods in their investigations of the thorax and its contents, were not to be deterred by its anatomic boundaries. Below the diaphragm, as well, there was visceral motion to be scrutinized. There, the organic structures were not endowed with sufficient natural contrasts of light and shade to make them visible; obviously, they must be made visible by artificial means, and, since the intra-abdominal portion of the alimentary tract, at least, was a structure in which motility was a very important phase of its physiology, it must be observed with the screen even as, in the thorax, the organs of respiration and blood-circulation had been observed.

It is of record that the earliest attempt to render visible by artificial means the course of the esophagus and the contour of the stomach was made by Strauss,[14] of Berlin, who, in 1896, used for this purpose gelatine capsules charged with reduced iron oxid and bismuth subnitrate. The shadows of these capsules upon his fluoroscopic screen were most indefinite and Strauss was evidently soon discouraged. He relinquished his investigations because, as he writes, "they appear to me to be of less practical importance than the question of changes in translucency of pathologic new-growth to the roentgen rays."

The promptly subsequent work of Walsh, Hemmeter, Naegele, Benedict, Williams, Lester Leonard, Cannon, and others, employing products of the mineral kingdom *not* administered with food, is well known, but even more familiar to us are the famous investigations of Cannon on the digestive functions of the cat, whereby alimentary roentgen diagnosis was christened in a bread-and-milk suspension of the subnitrate of bismuth, the ritual of the ceremony being interpreted through the eminently proper medium of the fluorescent screen.

Many of us did not then realize, perhaps, the significance of Cannon's work and the manner of its doing. Many, through the following years of the first fluoroscopic depression, as one might call it, were timorous of the threatened physical danger in routine fluoroscopic procedure. On the other hand, many were smitten, almost overcome, by the charm of the rapidly developing attractions of radiography, that fascinating but occasionally disappointing damsel our Bavarian colleague, Rieder, in 1904, had taught us to woo. Up to the day of Rieder's epochal accomplishment with reference to alimentary topography and physiology, efforts at *depicting* the contours of these viscera were largely confined to tracings and to punctate outlinings, both of them fluoroscopic methods. Rieder's results served as immense stimulation to others—most widely, perhaps, in Europe, but also here as shown in the early work of Hulst in 1905, Pancoast (with others) in 1906, and Pfahler in 1907. In short, American skiagraphy of the alimentary tract was merely keeping pace with the steadily increasing excellence of our general roentgenography. Although the technical quality of the radiography of the European school was of notable merit, by 1910 we were abreast of it, thanks to the high standard set by Caldwell and other advocates of fastidious technic, and, within a short time, ahead of it.

Undoubtedly, this radiographic development of ours had continued for some years at the expense of our roentgenoscopy. It was probably an excellent thing for both the European and American schools of radiologic practice that we were visited in 1910 by Haenisch of Hamburg, then the associate of Professor Albers-Schoenberg, as an official delegate to our national society meeting. After the meeting he visited many of our individual laboratories. Writing, later, in the German *Fortschritte* of his impressions, he said, in part: "We were surprised to find how little use is made of roentgenoscopy. The unfortunate accidents which followed the excessive use of the fluoroscope in the early days of roentgenology have brought radioscopy into disrepute [in America] and only of late is this method beginning to be introduced by certain practitioners who have travelled in Europe and are convinced that for certain cases screen examination is absolutely essential. . . ."

"Certain practitioners," thus referred to, made up a group of which Pfahler, Williams, Hulst, Skinner, and others were ardent members, and as a whole the report of Haenisch described with reasonable accuracy the setting of the American radiologic stage at the moment of the entrance upon the scene of one who, by force of his example and the conviction created by his writings, aided mightily in putting to an end the first American fluoroscopic depression—Russell Carman.

The course of Carman's professional life as a roentgenologist may, as we know, be divided into his shorter Saint Louis period and his thirteen fruitful years at Rochester, where he labored so diligently and so effectively. At the beginning of 1913, the resources in material he found at the Mayo Clinic served immediately to stimulate his predilection for gastrointestinal investigation and the practice of roentgenoscopy it involved, of which the results of his work in Saint Louis had already given indication. He found at Rochester the sympathetic yet reasonably critical attitude of one's colleagues that means so much to the pioneer in any form of specialized endeavor, and which stimulates his coöperative activity to the same degree that colorless assenting agreement retards it. The practice of roentgenoscopy is essentially a matter of close relationship between practitioner and patient, permitting of no delegation to subordinate hands. Therein lies its purity as a true Æsculapian ministration. Thus, at Rochester, it was Carman's happy lot to remain what he had set out to be—a physician rather than a scientist of the laboratory; for, however we may misapply in the name of convenience, the noun "laboratory" in speaking of the seat of radiologic practice, or, for the same reason, carelessly use the term "clinician" in contradistinction to the status of the radiologist, the fact remains that radiology is and

ever will be a clinical branch of the practice of Medicine.

It were impossible to amplify the true words already spoken, under this Lectureship, by Doctor Moore concerning the accomplishments of his preceptor, and the way in which they should influence the conduct of him who would undertake to solve the problems of roentgenology in abdominal diagnosis, but no consideration of the growth of American fluoroscopy could even approach completeness without a brief review of Carman's profound and salutary influence on it.

There is ample evidence in his writings to show that from the first moment of his radiologic activity he held the early investigations of Cannon in high appreciation. The fact that this work was based on the observation of visceral motion appealed to his sense of the importance of elucidating the presence of alimentary disease through the scrutiny of departures from the normal physiology. Thus he was moved to deplore the evident lack of early American interest in the future outcome of Cannon's contribution. It was over twenty-two years ago, as we were recovering from our first fluoroscopic depression, that Carman wrote:[11]

"It is to be regretted that a method which gives promise of such an advance in methods of gastrointestinal diagnosis, and which owes its inception to an American, Cannon of Boston, should have awakened in this country as yet so little interest as compared to that evinced by German and English clinicians and roentgenologists."

Carman's genius for roentgenoscopy was almost intuitive. Well before 1913, when one may say his broad contact with alimentary disease began, it had made itself evident. It was in 1910 —the year, in fact, of Haenisch's report on the meager use of fluoroscopy in the United States— that Carman thus expressed himself, in dealing with the subject of medical roentgenology as a specialty:[1]

"Roentgenoscopy deserves separate and praiseworthy mention. By the use of the fluoroscope and its modifications, the diagraphoscope and the orthodiagraph, physiologic and pathologic processes may be observed and studied *intra vitam*. Many of the phenomena of the circulation, of respiration, of digestion, can be seen in action. The progress of a peristaltic wave in the stomach, the pulsation of an aneurysm, the splashing of a pleural effusion, can all be viewed directly. *These must be seen on the screen, and not on the plate, to be of any direct value in diagnosis*"

And again, the following year:[2]

". . . it is not extreme to say that no x-ray examination of the chest or abdomen is complete without the use of the fluoroscope, and it should precede skiagraphy. . . . Upon the screen, living dynamic pathology is seen. On the other hand, the plate is a record of a single phase of conditions, which may be 'accidental or improvised,' as Holzknecht says."

These trenchant statements, made twenty-five years ago, may well be interpreted as Carman's confession of faith in the value of a procedure that had impressed him deeply from the early days of his medical career and which he applied consistently during the remainder of his life. As the immediate result of this application was formed the first expression of what may be termed the Carmanian influence on the present status of American roentgenoscopy. It is conveyed by these words:[2]

"Emphasis must be laid upon the necessity of observers remaining in the dark for at least twenty minutes before beginning the examination. The eyes then become accustomed and details, which otherwise would be lost, are plainly seen."

This point, small as it seems to be now that a world-wide fluoroscopic routine is established, was then of the greatest importance, and one which Carman iterated frequently; so, in later years, when it became generally known that this pupillary preparation was a part of the Mayo practice, and, since suggestions *ex cathedra* usually carry more weight, one's clinical colleagues elsewhere, perhaps acutely concerned in a given fluoroscopic problem, became less impatient of what had seemed to them a silly and unnecessary delay, and less expectant of a brilliantly detailed screen image the instant they entered the realm of roentgenoscopy from the bright light outside it.

At the outset of his day's screening, Carman had a most palatable remedy for impatience of this sort. Who of us oldsters does not remember those preliminary moments of general conversation in the darkness of the screening-room at Rochester, as we waited for our pupils to dilate —pupils ourselves, for the moment, of a great fluoroscopist, and in the very pleasant meantime dilating freely on every conversational subject, *except* radiology, from motor cars down?

There can be no doubt of the weight of Carman's diagnostic work, especially his gastrointestinal roentgenoscopy, as affecting the readjust-

ment of our statistics relative to the incidence of duodenal ulcer. In 1914, in one of his many discussions on the subject, he took occasion to raise a collateral point which has subsequently done much to establish closer *liaison* between hospital roentgen departments and other clinical services, surgery in particular. Said he:[4]

"Notwithstanding the constantly growing mass of convincing statistics, many clinicians and especially a few gastroenterologists of this country and continental Europe are on record as claiming that duodenal ulcer is a lesion which rarely occurs. Their inability to recognize its frequency is probably due, as W. J. Mayo states, to the presence of the abdominal wall. Speaking from a radiologic point of view, I might say that during the last year only have I come to realize the frequency of this condition through following cases to operation and looking into the abdominal cavity with the surgeon. I would certainly recommend this procedure to those that are skeptical, because a large number of the patients treated for 'chronic dyspepsia' have duodenal ulcer. . . ."

That "year" to which Carman refers—his "year of realization," as it were—was his first year at Rochester. It is probable that, even at that early day, he carried with him to the operating room well thought-out diagnostic information which, more often than seldom, it was merely for surgery to corroborate. But what soon became apparent was the *spirit* in which he invariably approached the test of surgical revelation. Counterevidence or disproof, when it was forthcoming, he accepted in the acquiescence of the true scientist, nay, he welcomed it with the warm partisanship of the humanitarian if the findings redounded to the advantage of the patient. Carman early formed this habit of operating room attendance, and its obvious advantages to both surgeon and roentgenologist soon became generally recognized. Its constructive effect on the hospital roentgenologic units of the country was the creation of a spirit of thorough interdepartmental understanding and coördination through opportunity for truly clinical comparison of evidence. Who shall say that this spirit, now so generally manifested everywhere, is not another expression of the Carmanian influence on the status of American roentgenoscopy?

From Carman's habit of establishing and fostering such close clinical association with his colleagues, results of combined observation of the greatest worth were the inevitable outcome. Attesting to this is the exceedingly important communication of Carman and Balfour,[6] in 1915, in the matter of gastrojejunal ulcer. Up to that moment, consideration of this lesion had been preponderantly surgical, and roentgen-ray evidence in diagnosis, when utilized, had been inconclusive. Doctor Moore, from this rostrum last year, mentioned the far-reaching results of this report; for our consideration today be it said that the fourteen cases by way of illustration were accurately and in detail described on a basis of initial fluoroscopy. "Roentgenoscopy," writes Carman in this paper, "is particularly necessary in examinations for gastrojejunal ulcer," and proceeds, by the conclusions of the authors, to prove the truth of a statement that has since become an axiom. From the purely historical viewpoint this paper is equally important, for Carman's share in it gave him the opportunity to pour oil on the waters of controversy that were already becoming turbulent. His words reveal the nature of the discord:

". . . In every examination of the digestive tract both roentgenoscopy and roentgenography should be employed. They should not be regarded as independent competitive methods, but as complementary parts of one method, for exclusive reliance upon either alone is likely to result in overlooking some important feature of a case. . . ."

Carman had no taste for petty polemics. "Controversies, sometimes heated, but Pickwickian," as he once wittily classified them, were against his very nature, but in maintaining the larger defences of his position he was strong. It was this strength, added to the prominence of his station in American Medicine, that made his the protagonistic key-position in what has been loosely termed the "fluoroscopic school of thought" in roentgen diagnosis in this country, especially as applied to the digestive tract at the moment of which we are now speaking. This school of thought, or, more properly, of practice, "was an evolutionary product of the so-called Continental doctrine, in that its mode of investigation was basically roentgenoscopic, formulating its diagnosis, after the precepts of Holzknecht, on a factorial consideration of the 'symptom-complex' presenting in the given problem, as well as upon its radiographic records in confirmation." These principles had their advocates in men of such strength as Forssell, Haenisch, and Barclay and, in this country, among others, Crane, Hulst, Pfahler, Case, and Skinner.

Carman's breadth of mind embraced all phases of his work and permitted of no bigotry as to the infallibility of his theory and practice. His readiness to admit the possibility of their points of weakness or doubt seemed to amount almost to an obsession. He wrote entire papers on the limitations of roentgen diagnosis, but in the furtherance of any tenet that he had proven to be sound, he was as firm as bed-rock. The fact that the findings of roentgenoscopy played so important a part in the procurement of the data that gave to the work of Carman and Balfour its great value, together with the fact that this work showed conclusively that accurate and detailed circumstomal roentgen diagnosis may spell the difference between postoperative comfort and postoperative misery that "maketh the heart sick," would seem to indicate, it is believed, that herein lies yet another expression of the salutary Carmanian influence on the development of fluoroscopy.

By his display of common sense, Carman had done much, as has been said, to allay the growing fear of fluoroscopy which he found to exist when he entered the practice of radiology, and somewhat later, during the first year of his Rochester period, or, to be more exact, twenty-two years ago, in this city, in this month of June, by means of a paper describing his technical methods,[3] he gave voice to his views on the fluoroscopic depression brought about by an hysterical timidity that had taken the place of sensible caution. To those about to discard their fluoroscopic apparatus he suggested the exercise of temperance rather than prohibition. "I would not urge any roentgenologist," he said, "to take risks which to him seem imprudent and which might possibly result in his injury, but candor compels me to express my very sincere belief that we have gone from one extreme to the other, from extraordinary and ignorant rashness to extraordinary and unnecessary caution." He deplored the influence of this unnecessary caution as was shown in the heavy and unwieldy construction of much of the fluoroscopic apparatus of that day. Since this ponderous construction prevented effective access to the patient for purposes of palpation, Carman reasonably complained that "the screen examination, thus 'denatured,' loses half its interest and value." Those who were screening before Carman's day, or even contemporaneously with his earliest gastrointestinal practice, will be prepared to agree that this protest of his against such Gargantuan architecture in screening apparatus whereby, amid its pillars and flying buttresses, the person of the unprotesting patient became effectually shielded from all efforts of the palpating hand, was the beginning of a reactionary period of simplicity in the construction of these devices—another manifestation of the "Carmanian influence," but perhaps of lesser importance than many others, in spite of its specific value.

But this reaction in the direction of increased simplicity, while it had its permanent good effect on after years, became much too pronounced under the stress of the urgencies of the Great War. Roentgenoscopic practice constituted almost ninety per cent of the military radiologic activity in intermediate areas behind the dressing station zone and as far back as the base hospital. By the very simplicity of its construction, much of the apparatus in such areas was woefully inadequate as far as protection of the operator was concerned. At casualty clearing stations or evacuation hospitals, screening apparatus was necessarily of simple installation with a view to its quick removal in case of forced retirement.

Not many months after the close of the War, possible results of over-exposure to x-rays in the Service and otherwise were being noted in the occurrence of aplastic anemia and other blood dyscrasias. Perhaps because of the subtlety of these effects, perhaps by reason of wider publicity, greater agitation was created than had been the case fifteen years before. The second fluoroscopic depression was on; many relinquished completely the use of the screen, and roentgenoscopy seemed about to receive another periocular contusion, as it were. Russell Carman, however, from the stronghold of his now eminent position, treated its prodromal signs, as he had done previously, with the cold compress of common sense. His temperate attitude was matched by that of others of his peers who realized that any curtailment of the growing beneficence of roentgenoscopy was unthinkable, and that radiation-danger, not only to its devotees but in any form of radiologic practice, could be controlled only by concerted regulation of the element of protection, and not by haphazard effort. Accordingly, in 1920, the American Roentgen Ray Society formed its standing committee for roentgen-ray and radium protec-

tion. The movement was soon taken up by similar societies in Great Britain, and there had the strong and influential support of the National Physical Laboratory to a degree not as promptly reached by our National Bureau of Standards. The subsequent development of this activity is familiar to all; through Continental coöperation there has been created the International Committee on X-ray and Radium Protection, and among us there has appeared the Bureau of Standards Handbook on Roentgen-ray Protection.

In spite of these organized precautionary measures, to which the registered American radiologist must subscribe and comply, and thus may practise his profession with absolute safety to his patient and relative safety to himself, the untoward potentialities of uncontrolled fluoroscopic practice are still with us. There yet endures the unprincipled charlatan who, within the mystifying aura of the roentgenoscope, seeks to hoodwink his trusting victim as he shamelessly lies into credulous ears as to what the luminous screen reveals. We still have the manufacturer who is so far unmindful of the respect of the qualified radiologist that he continues to persuade the internist or the general practitioner of the advantage in the possession of a fluoroscope as a bit of decorative office furniture, which it is ultimately to become after producing its yield of disappointment. Also, there is the less serious matter of the commercially wolfish shoe dealer garbed in the sheep's clothing of science, as he asks us to enjoy, with him, by fluoroscope, the sublime vision of our *hallux valgus* which he proposes to fit, plus or minus, into the tight leather of vanity. Roentgenoscopy, with all our modern devices for protection of the operator, is still, in unpractised hands, a procedure potentially serious, to use no stronger an adjective. So is any manipulative undertaking wherein are combined the "personal equation" and a complex distribution of high-voltage electricity with the radiation phenomena attending it. The laws of physics may be impeccable, but human agencies are defective. Hence the value and applicability of the fluoroscopic observations of Barclay, of the University of Cambridge, or the more recent researches of the Americans Cilley, Leddy, and Kirklin.

During the later years of Carman's life, two events occurred which must have been to him exceptional sources of satisfaction. One of them was the amplification by Forssell and Åkerlund of Forssell's early work of 1912 on relief-demonstration of the alimentary mucosal folds. In this later elaboration, Carman could see developed one of the first manipulative adjuncts to fluoroscopy which he had practised and taught—screen observation under applied pressure; not mechanical pressure by cones or cylinders or balloons that since may have added to the accuracy of these observations, but pressure of his good right arm, with a "palpatory eye" at the tip of each finger! Carman's physique afforded him aid that no man of lesser build could hope for.

The other satisfaction must have been the fruition of the work which Moore authoritatively declares to be the great contribution of Carman's life—the discovery of the "meniscus sign" of ulcerating gastric cancer. With what pride Carman states in his report[5] that fluoroscopy is essential for the demonstration of this lesion, because manipulation is nearly always requisite for its exhibition—such was his fascination by the rewards of palpatory effort!

Radiologists of Minnesota: To you especially has been given a great heritage. How well, already, you have utilized this heritage may be seen from the quality of the research that has resulted from your energies. Of this none can speak more truly than one who has scrutinized of late the literature of the science of diagnostic radiology. Continue to hold fast and be true to the principles inherent in this bequest. Be rejoiced in your power to pursue the art of fluoroscopy in its Carmanian sense, which exemplifies the true and hallowed relation between physician and patient. Apply that power with the sympathy and the understanding that was his.

Friends and allies of radiology in other special fields or in general medicine: It cannot be difficult for you to appreciate how close a bond between us has been woven by the natural gifts of a man whose useful life has been so influential in converting into a clinical art the applications of a natural phenomenon which forty years ago was revealed to the gentle Professor of Physics in that modest laboratory at Würzburg. May this bond serve further to interlock securely, and ever in the interest of those in need of succor, the fine traditions of your calling and the high principles of practice laid down by Carman and

now carried out by his former colleagues and disciples throughout your state of Minnesota.

Together we must lift high the lamp of Learning. The higher we raise it, the more widely will fall the rays of its glowing flame, its sustaining essence refined and purified by the spirit of such as Russell Carman!

References

1. Carman, R. D.: Medical roentgenology as a specialty. Jour. Missouri State Med. Assn., 7:121, 1910.
2. Carman, R. D.: The diagraphoscope. St. Louis Med. Rev., 60: (5 n. s.) 198, 1911.
3. Carman, R. D.: The technic of roentgen-ray examinations of the gastro-intestinal tract, and the interpretation of the screen and plate findings. Jour. A. M. A., 61:321, 1913.
4. Carman, R. D.: Radiologic signs of duodenal ulcer, with especial reference to gastric hyperperistalsis. Jour. A. M. A., 62:980, 1914.
5. Carman, R. D.: A new roentgen-ray sign of ulcerating gastric cancer. Jour. A. M. A., 77:990, 1921.
6. Carman, R. D., and Balfour, D. C.: Gastrojejunal ulcers; their roentgenologic and surgical aspects. Jour. A. M. A., 65:227, 1915.
7. Crane, A. W.: Skiagraphy of the respiratory organs. Phila. Monthly Med. Jour., 1:154, 1899.
8. Edison, Thomas A.: Quoted in American Journal of Photography, 27:160, 1896.
9. Glasser, Otto: Wilhelm Conrad Röntgen and the Early History of the Roentgen Rays. Springfield (Ill.) and Baltimore: C. C. Thomas, 1934.
10. Hammer, E. W.: Trans. Am. Institute Electrical Engineers, 1903.
11. Mills, R. W., and Carman, R. D.: The x-ray in the diagnosis of gastric ulcer and its sequelæ. Surg., Gynec. and Obstet., 17:1, 1913.
12. Pfahler, G. E.: Roentgen diagnosis of diseases of the lungs. Jour. A. M. A., 46:23, 1906.
13. Salvioni, E.: Proc. Accademia Medico-Chirurgica di Perugia, 8: No. 1-2, (Feb. 8) 1896.
14. Strauss: Beitrag sur Wurdigung der diagnostischen Bedeutung der Röntgen-durchleuchtung. Deutsche Med. Wchnschr., 22:161, 1896.
15. Williams, F. H.: The uses of the fluoroscope. Communications of the Massachusetts Medical Society, 17:858, 1896-98.
16. Williams, F. H.: Quoted by Crane. Loc. cit. ref. 7.

THE PROGNOSIS AND TREATMENT OF CORONARY OCCLUSION*

WILLIAM S. MIDDLETON, M.D.

Madison, Wisconsin

"And he will manage the cure best who has foreseen what is to happen from the present state of matters."—HIPPOCRATES, *The Book of Prognostics.*

THE natural evolution of the knowledge of disease has usually been from diagnosis to therapy and prognosis. The accurate clinico-pathological correlation of disease processes was the great contribution of the French school of the late eighteenth and early nineteenth centuries. Nor has the necessity for this function passed. Bacteriology gave a tremendous impetus to this growth and by establishing the etiology of the several infectious diseases not only facilitated their diagnosis but also paved the way for the use of specific biologic agents in their prophylaxis and treatment. The application of physiological and biochemical methods to the study of clinical problems has opened a new vista in the early and the more complete appreciation of disease. Still, the clinical recognition of a disease will anticipate the consideration of its prognosis and treatment in most instances.

Coronary occlusion is no exception to this general rule. The first intra-vitam recognition of coronary thrombosis was reported by Hammer[30] in 1878. The classical clinico-pathological consideration of the subject by Dock[16] followed eighteen years later. Still no general interest appears to have been aroused in the matter until its clinical implications were driven home by Obratzow and Strachesko[62] and by Herrick[31] in 1910 and 1912, respectively. The triad of subjective manifestations suggested by Obratzow and Strachesko, status anginosus, status dyspnœicus and status gastralgicus, satisfied the clinical requirements of clarity, brevity and applicability. In the intervening years the picture has been amplified, so that today the bedside recog-

*From the Department of Medicine, University of Wisconsin.

nition of coronary occlusion is a commonplace.

Diagnosis held sway in all of the earlier discussions of coronary occlusion. True, Herrick[31] suggested the possibility of survival of certain patients after this accident; but the prognosis had little place in the literature of the subject until the past decade. Since then Levine,[45] Herrick,[32] Christian,[12] Pardee,[65] Jegorow,[38] Luten,[52] Burton and his fellow workers,[11] Evans[18] and others stimulated interest in the prognosis of coronary occlusion by recitals of recovery. Comprehensive studies of this phase of the subject by White,[92] Parkinson and Bedford,[66] Levine,[46] Conner and Holt,[13] and White and Bland[94] have served to extend the clinical interest in the prognosis and treatment of coronary occlusion.

An adequate appreciation of the possibility of survival after occlusion of a coronary arterial branch depends upon an understanding of the basic anatomic facts and of pathologic principles as applied to this system of vessels. Gross[27] and Spalteholz[83] have evolved masterly descriptions of the blood supply of the myocardium. Briefly stated,[27] the right coronary artery is responsible for the nutrition of the entire right ventricle with the exception of the left third of its anterior wall, where the branches of the left coronary artery extend. The left coronary artery supplies the anterior wall of the left ventricle and the left half of its posterior wall, whereas the right coronary artery affords nutrition to the right half of the posterior wall of this chamber. The left coronary artery supplies the anterior portion of the interventricular septum, and the right coronary the posterior portion. A dual supply may be anticipated in the zones of approximation of these independent sources of nutrition. Sixty per cent of the studied hearts derived the blood supply for the sino-auricular node from the right coronary artery, while the auriculo-ventricular node was supplied by the right coronary artery in 92 per cent of the studied cases. The right limb of the auriculo-ventricular bundle is nourished by the left coronary artery, and the left limb by branches of both coronary arteries. It is impossible to generalize the distribution of the coronary vessels to the auricles.

From a prognostic standpoint the question of anastomoses assumes a vital importance in event of a coronary occlusion. Without a reasonably adequate collateral blood supply, occlusion of a coronary arterial branch of any size must inevitably terminate fatally. The earlier experimental studies of Porter[68] upon the effects of ligation of the coronary arteries supported the view that cardiac arrest bore a direct relation to the caliber of the coronary vessel ligated and such anastomoses as existed were inadequate for collateral circulation. Miller and Matthews[58] found that successive ligations of major branches of the left coronary artery ended fatally, but the ligation of either of its main branches was attended by cardiac failure only after 1 to 3 months. Fortunately in the interest of clarity the Weigert-Cohnheim controversy has finally been settled through the definition of anastomoses between the coronary vessels by Hirsch and Splateholz.[35] Wearn and his fellows[37,89,90] have contributed extensively to the knowledge of this detail. Small but widespread communications with branches of the internal mammary artery and anterior branches of the thoracic aorta (mediastinal, pericardial, bronchial, superior phrenic, intercostal and esophageal arteries) were established by injection studies of the coronary vessels. These anastomoses were abundant through the arteriæ telæ adiposæ and about the great vessels leaving and entering the heart, those about the pulmonary veins being especially notable. More significant perhaps have been their observations relative to the nutrient possibilities of the vessels of Thebesius in event of coronary occlusion. To these vessels, which they divided into the arterio-luminal vessels, arterio-sinusoidal vessels, and myocardial sinusoids, they attribute a major place in replacement of the coronary arterial function after extensive occlusion. Grant and Viko[26] limited the connections between the coronary arteries and the Thebesian vessels to channels of capillary dimensions. Furthermore, Stella[84] maintained that blood flows through these vessels only in diastole and by inference minimized their functional importance. Against this adverse evidence may be arrayed a growing series of survivals after occlusion of both major coronary arteries.[7,44,75,78] A further collateral source of nutrition was suggested by Thorel[85] in the vascularization of the impoverished myocardium in case of coronary occlusion through pericardial adhesions. Moritz, Hudson and Orgain's[59] injection studies of four hearts, the seat of extensive pericardial adhesions but without coronary disease,

confirmed this position by establishing widespread anastomoses of the coronary arteries with the internal mammary arteries and anterior branches of the aorta through such adhesions.

As early as 1880 Weigert[91] suggested a possible protective mechanism on the part of nature in providing collateral paths of nutrition in case of coronary encroachment. Dock[16] subscribed to the same view. Gross[27] proved that such pathways became more abundant and adequate with advancing years. Galli[23] placed this matter on a sound footing by proving that gradual occlusion of the branches of the coronary arteries was a vital factor in assuring ample anastomoses. Finally, Oberhelman and Le Count[61] by injection studies of diseased and normal hearts found adequate collateral circulation only in those with antecedent coronary sclerosis. In the absence of such sclerotic changes either a total lack or an inadequacy of such anastomotic channels was observed.

The natural implication from such observations[61] was that the outcome of coronary occlusion depended upon three important factors, namely the size and importance of the obstructed artery, pre-existent channels and time to render such anastomoses effective. Coronary occlusion may develop gradually or suddenly. If embolism be omitted from the present consideration by reason of its infrequence and special implications, coronary sclerosis is the common background of the acute and the chronic types of occlusion. Von Leyden[87] thus classified the changes incident to coronary obstruction:

1. Sclerosis, without myocardial change which presupposes an intact nutrition until sudden occlusion.
2. Infarction with myomalacia cordis.
3. Slower occlusion with myofibrosis which may occur diffusely or in patches and which may result in aneurysm of the heart.
4. Combinations of these types.

With myocardial infarction there occurs quite commonly a reactive pericarditis. An effusion may result or adhesions between the two layers of the pericardium may afford an appreciable support and at the same time lend an unusual source of nutrition to the impoverished myocardium.[85] Eventually such adhesions may increase the work of the heart. Within the chambers of the heart overlying the areas of infarction, especially in event of aneurysmal dilatation, mural thrombi may form carrying with them the threat of detached particles which may act as remote emboli. Acute coronary occlusion from a septic embolus may lead to abscess of the myocardium. Otherwise the pathologic sequences from coronary embolism parallel in general those of thrombosis.

So intimately is the fate of the victim of coronary occlusion tied up with the sequences of events in the affected myocardium and the time relationships incident thereto that attention is next directed to these details. Obviously an artery of a very small size or with an adequate collateral relationship, may be occluded without serious consequence and perhaps without clinical evidence of its occurrence. Admitting appreciable encroachment upon the nutrition of a segment of the heart wall, in only a minority of cases may one hope for a relatively complete restoration of an adequate blood supply and escape from structural changes through full anastomotic branches or canalization of the occluding thrombus. The area of infarction is invariably smaller than the actual distribution of the occluded artery. The anterior apical portion of left ventricle supplied by the descendens branch of the left coronary artery is only slightly more frequently obstructed than is the posterior basal portion of the left ventricle supplied by the right coronary artery.[5] Levine[46] summarized the pathological changes in the myocardium incident to coronary thrombosis in this manner: Edema and hemorrhagic extravasation characterize the first three or four days; then necrosis dominates the picture from the fourth day to the end of the third week; finally, repair, which is remarked as early as the sixth or seventh day, does not become a striking feature until after the third week and does not appear adequate until more than five weeks have elapsed. The infarcted zone of the myocardium may eventually be the seat of dense calcium deposits.[15,36] These pathological observations have an extremely important clinical bearing upon the prognosis and treatment.

Death from coronary occlusion may result from one of a series of causes. Fulton[22] recorded death from embolism, myocardial impoverishment, rupture of the heart and interference with conduction. It is generally admitted that a considerable percentage of patients succumb to attacks of acute coronary occlusion before

medical attention can be had. In this group still fall the dwindling cases of so-called acute indigestion, for the elucidation of which students of coronary disease have done so much. Levine[46] pointed out the frequent responsibility of ventricular fibrillation for this manner of death from coronary occlusion. As may be gathered from a perusal of the pathologic sequences in an area of myocardial infarction, the fourth to the fifteenth day is the period of most serious threat from rupture of the heart. At any time in the first three weeks the patient may die from embolism to the brain, or to other important organs. After the third week the patient is relatively safe on this score. Having escaped these more acute manners of exodus, the myocardium may gradually become incompetent and the classical symptoms of congestive failure supervene after a period of weeks or months.

Willius[95] epitomized the situation by stating that one-half of the cases of coronary occlusion die at the time of the attack or in a few days. The survivors live a few months to several years, with varying degrees of cardiac handicap. White,[93] on the other hand, related an extraordinary example of longevity after coronary occlusion. His patient, a man of sixty-three years at the time of the attack, resumed his normal activities after several months of rest. Ten years after the coronary episode he indulged in strenuous mountain-climbing without circulatory embarrassment. Death at the age of eighty years was occasioned by apoplexy and at necropsy the heart revealed essentially only the dense scar of the healed infarct. Parkinson and Bedford[66] were able to trace the course of 99 of their 100 patients with coronary thrombosis. Of the surviving 68, the duration of life to the time of the study exceeded 5 years in 2, ranged from 2 to 5 years in 9, 1 to 2 years in 19 and up to 1 year in 38. The obvious criticism of a wide scatter of insufficient data detracts from this report. Then, too, the length of time covered is very short. In Levine's group of 143 cases[46] the immediate mortality was 53 per cent. The duration of life in the survivors from the acute episode (42 still living) was twenty-four months. The series of Conner and Holt[13] revealed an immediate mortality of 16.2 per cent. Of the surviving 117 patients, 75 per cent were in good health at the end of one year; 56 per cent at the end of two years; 21 per cent at the end of five years, and 3.4 per cent after ten years. Of the patients having a single attack one-half are living and one-third are in good health. A single patient remained in good health for seventeen years and died in a second attack of coronary thrombosis eighteen years after the first episode. In 1931, White and Bland[94] reviewed their series of 200 cases of coronary thrombosis. Of this group, 101 patients died an average of 1.5 years after the accident. The duration of life for the entire 200 patients was 2.4 years at the time of the report. A patient with coronary thrombosis reported before the Minnesota State Medical Association in 1927[56] is still alive and active nine years after his accident. Although he plays eighteen holes of golf each day without the slightest discomfort, there is some dyspnea on mounting a flight of stairs. The electrocardiogram shows rather low voltage (6 mm.) and there is a perceptible elevation of ST_2 as the sole remnants of a classical T type of change from infarction of the anterior wall of the left ventricle.[6,67] Fluoroscopy revealed an adhesion between the tip of the left ventricle and the parietal pericardium. In a recent report Cooksey[14] found 78.1 per cent of thirty-two survivors from coronary occlusion restored in such measure as to permit of a complete resumption of normal activities.

The prognosis of coronary thrombosis is admittedly very difficult and a reflection upon the numerous cited variables offers a partial explanation of the problem. It is moreover a strictly individualized question and generalities must be viewed with this reservation always in mind. Age is an important factor in prognosis. While advancing years and sclerosis prepare the collateral channels against the accident of coronary occlusion, there is always the probability of a concomitant myocardial malnutrition and resultant fibrosis from these sources. The general experience gives a more favorable prognosis to the younger subject with antecedent sclerosis. For example Levine[46] found the average age of his surviving patients to be 54.7 years as compared with the 61 years of the fatal group. Having recovered from the immediate episode it is significant that the younger individual more regularly resumes a fuller share of his previous activities.[14,46] Although coronary occlusion is much more frequent in men than in women, this circumstance apparently has no

bearing upon the prognosis. An analysis of forty-four cases from the Wisconsin General Hospital substantiates these details. Likewise as reported by Levine[46] and Conner and Holt[13] our series shows no apparent discrepancies among the diabetics and hypertensives as compared with the general group. Syphilis was a predisposing factor in only one of our group in whom death occurred eight months after thrombosis of the right coronary artery with infarction of the posterior basal portion of the left ventricle.

Pain, the most striking subjective manifestation of the coronary occlusion as a rule, may be a useful prognostic sign or it may be completely misleading. Conner and Holt[13] found some correlation between the severity of the pain and the gravity of the outlook; in general the more severe the pain, the higher the mortality. On the other hand, their figures afford enough exceptions to militate against a wide acceptance of this criterion in prognosis. In our experience some of the most ominous attacks are either mild or free from pain. Angina sine dolore may occur independently of an altered rhythm and is a symptom-complex of unusually grave portent in coronary occlusion. Unquestionably certain cases so designated may, as Wayne and Graybill[88] reported, depend upon disturbed rhythm; but Herrick[33] and others have called attention to so-called painless attacks of coronary occlusion. Obratzow and Strachesko[62] denominated the suffocative dyspnea as a pain equivalent and, as Herrick[33] pointed out, the rapid fall in blood pressure may be a further substitution or replacement phenomenon.

One notable instance of this order in our group is recalled to mind. The patient, a white male sixty-four years of age, was accustomed to run short distances to or from work when his tasks as a janitor did not afford adequate exercise. Without warning he was stricken with dull pain beneath the lower sternum. This pain became progressively worse, to assume overwhelming proportions; whereupon he fainted. Upon admittance to the hospital he was in profound shock and the blood pressure on frequent determinations through this day ranged from 65 to 90 mm. Hg. systolic. Thereafter, on three occasions one to four days later, the patient experienced "sinking" spells or attacks of weakness in which the systolic blood pressure fell from slightly over 100 mm. Hg. to 80 or 90 mm. Restlessness and apprehension were conspicuous concomitants of each of these episodes and a leaky, clammy skin with gray pallor was regularly present at such times. The electrocardiograms were characteristic of infarction of the anterior wall of the left ventricle.[6,67] A retrograde propagated thrombus starting from the initial site in the descendens branch of the left coronary artery and involving the orifices of successive branches of this vessel was postulated in explanation of the striking clinical course. The patient succumbed on the fifth day after the coronary accident from rupture of the left ventricle. The original thrombosis had occurred rather low in the descendens branch of the left coronary artery and rupture of the heart had taken place through the necrotic infarct; but of particular interest was the ready tracing of the thrombosis propagated upward to involve a sequence of the larger branches of the left descendens artery. In concluding this detail it should be emphasized that, important though pain may be in a prognostic relation, its absence or the occurrence of equivalents in respiratory and circulatory symptoms should always be considered an ill omen.

The mental status of the patient with coronary occlusion takes unusual prominence in evaluating the prognosis. Early apprehension is the rule. Then having weathered the initial storm the physician must be alert to the reaction. Too frequently the fear of the early phase gives way to a remarkable euphoria. This state of mind is importantly controlled from the standpoint of treatment; but it must never be misleading, since it is often the calm before the delayed storm. Not infrequently the mental anxiety persists and one is taxed to learn whether it is cause or effect. Certainly it is not unusual to have recurrent thrombosis in this group of patients. Delusionary, hallucinatory and illusionary states may complicate the mental picture and render the outlook more dangerous. Unconsciousness from the outset gives a particularly bad prognosis.

The curve of the arterial blood pressure is one of the most important criteria of the clinical course and the outcome of coronary occlusion. In the first place it must be borne in mind that the fall in the blood pressure subsequent to coronary occlusion is relative. Accordingly a hypertensive subject may have levels of blood pressure persistently above normal but at a distinctly sub-

natural level for himself after this accident. Not only is the degree significant in this relation, but the maintenance of a low or falling arterial pressure thereafter is particularly ominous. The duration of the relative or absolute arterial hypotension is an important index of the extent of myocardial damage, although survival and a resumption of a reasonable measure of physical activity may occur in individuals where very low pressures have been recorded for weeks after the occlusion. Of course, this tendency is not without its compensations, since Levine,[45] Herrick[32] and others have stressed the disappearance of pain dependent upon the lowered blood pressure that may succeed coronary thrombosis. With the falling or maintained low arterial pressure may be anticipated a leaky clammy skin and a rapid thready pulse.

Turning to the further examination of the circulation, cyanosis and dyspnea arrest the attention. As pain equivalents their importance in prognosis has already been discussed; but as evidence of beginning or continued circulatory incompetency they are given further weight. Particularly is this true when there is added pulmonary edema. These signs may be paroxysmal or continued. The prognosis is much better in the former type; and if these manifestations be explained on the basis of left heart failure, their disappearance will be reassuring. Evidences of congestive failure may supervene; and, although commonly delayed, their occurrence spells the end of at least one episode of circulatory balance. Of course, decompensation from myocardial incompetency on a coronary background may be controlled repeatedly by proper measures in a given individual, but the margin of safety is narrow and the occasion of cardiac failure sooner or later arrives in such a subject when treatment is unavailing. A glance at the reserve force of such a heart from time to time may enable the clinician to forecast and even forestall too early an expenditure of the cardiac reserve.

The heart itself may give valuable prognostic information in the early phases of the story. Antecedent disease and cardiac hypertrophy may be taken as presumptive evidence of a poorer prospect. The cardiac tones have a definite place in forming this composite picture. They may be extremely feeble. Tick-tock and embryocardiac sounds are very bad omens. Withal a peculiar mushiness of muscular tones and the appearance of murmurs of relative valvular insufficiency should be given their due weight. Arrhythmias, subsequent to coronary occlusion, bear a serious significance. Particularly is this true of auricular fibrillation, flutter and the various types of heart block. The persistence of such irregularities is even more ominous than their transient incidence, although Levine[46] found no higher mortality in those patients having auricular fibrillation, extrasystoles or partial heart block subsequent to coronary occlusion. He felt that complete heart block and ventricular tachycardia probably carried greater threats to life. Contrary to Levine,[46] in our series the occurrence of a pericardial friction did not increase the gravity of the prognosis. Remote embolism, serious in itself as a basis for disability and death, carries the added implication of intracardiac thrombosis.

The constitutional reaction to the myocardial infarction varies widely in different individuals. Neither the degree nor the duration of the fever bears any fixed relation to the clinical course and the outcome. In general the higher and more continued the fever the more serious will be the prognosis. Libman's observation[50] of the leukocytic response to coronary thrombosis and infarction has been generally confirmed. The polymorphonuclear leukocytosis, which may anticipate the fever and occur independently of the reactive pericarditis, is apparently a more delicate index of the status of the infarction than is the fever. Accordingly close observations of the numerical and the differential white blood pictures may afford valuable information as to the course of the myocardial process. Isolated counts are inconclusive and even the more detailed studies lack finality. Libman[50] pointed out that no case of coronary thrombosis failed to show a leukocytosis which might begin as early as 1 hour and 45 minutes after the attack. In his judgment a long continued leukocytosis indicated either a pulmonary complication or an intraventricular thrombus.

The recent observations relative to the sedimentation rate of the erythrocytes in coronary thrombosis must be cited in this direction. In 1931 Wollheim[96] mentioned the increased sedimentation speed in this condition without further comment. Shortly thereafter Rabinovitz and his co-workers[70] remarked the appearance of this phenomenon somewhat later than the fever and the leukocytosis. Furthermore the speeding of

the sedimentation rate persisted after the subsidence of these two factors. In their judgment its determination served as a better index of the healing of the myomalacia than the fever or the

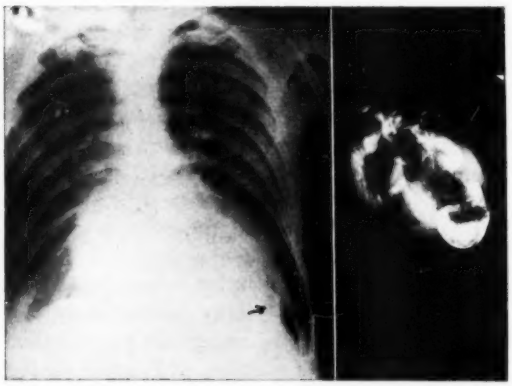

Fig. 1. Roentgenograms of chest and of heart (distorted somewhat by postmortem incisions) showing calcified infarct and intracardiac thrombus.

leukocytosis. They did not commit themselves to a parallelism between the degree of myocardial change and the sedimentation speed. Since then, Burak,[10] Singer[77] and Bickel and his fellows[5] have confirmed the regularity of its occurrence and have given it unusual prognostic significance. The last named concluded that while the sedimentation speed was not always proportional to the gravity of the pathologic process, if there were no infection, a more rapid rate of sedimentation always indicated a continued cellular disintegration. In their judgment it served as one of the most reliable indices of recovery.

Roentgenology affords information of value in the visualization of the size and the contour of the heart. Obviously progress studies from the time of the coronary accident (and if possible anticipating the same) would give valuable data relative to these details. Orthodiascopy has served a further function in delimiting areas of infarction by the same principle of inelasticity or failure to respond to the wave of cardiac contraction that is remarked in the indurated area of a peptic ulcer in the stomach failing to participate in the orderly gastric peristalsis when viewed under the fluoroscope. Localized pouching or bulges may fix the site of aneurysmal dilatations of the ventricles. Obviously the subject must be investigated in all positions to throw the infarcted area into proper silhouette. Occasionally calcium deposits in the devitalized heart muscle may be demonstrated through the characteristic density.[15,36] Four instances of this order have been encountered in our series (Fig. 1). Obviously these observations will more importantly afford an objective measure of the location and extent of the deformity than supply prognostic information.

From the early observations of Smith[79,80] upon the electrocardiographic results of ligating the coronary arteries of the dog, a world wide interest in the clinical application of the principles involved has led to many important contributions to the diagnostic features of the tracings in coronary thrombosis. A volume could be written on the subject and it has not yet been exhausted. The prognostic significance of the electrocardiographic changes has not been completely settled. If it be admitted that the characteristic electrocardiographic changes succeeding coronary occlusion are dependent upon myocardial injury, Oppenheimer and Rothschild[63] conclude that the prognosis would be better in event of a normal electrocardiogram. Extended experience has stressed the importance of repeated electrocardiographic studies in tracing the course and in attempting to fix a prognostic value upon certain changes in the constituent complexes. Transitions may be remarked from normality to pathologic configurations (Fig. 2). Then the recuperation may likewise be followed in sequence to or approaching the prethrombotic curves (Fig. 3). Pardee[64] assumed that the T wave deflection arose from the reaction of repair about the area of infarction. Parkinson and Bedford[67] in an illuminating study attributed the electrocardiographic changes, especially in the T waves, to edema and leukocytic infiltration after infarction. The RT deflection was ascribed to the frank necrosis of the heart muscle. Accordingly they assumed the subsidence of these deviations to depend upon recovery of myocardial function. Certainly they are observed to occur coincidently with the maintained recovery of the blood pressure and improved circulatory competency. Hamburger[29] concluded that the prognosis was not good if the T wave remained negative, or if the inverted T wave were associated with further evidence of widespread myocardial injury, such as arborization or bundle branch block. Conner and Holt[13] concurred in these

details. Barnes' recent analysis[4] was especially illuminating. He concluded that a low voltage in the ventricular complexes of all leads was a bad prognostic sign. In his extended experience the low voltage QRS waves occurred twice as frequently among those dying as among the survivors. Furthermore, typical T_1 and T_3 formulæ carried a better prognosis in that they appeared six times as commonly as the atypical curves in those recovering from the coronary accident. Barnes noted that atypical tracings were frequently associated with multiple in-

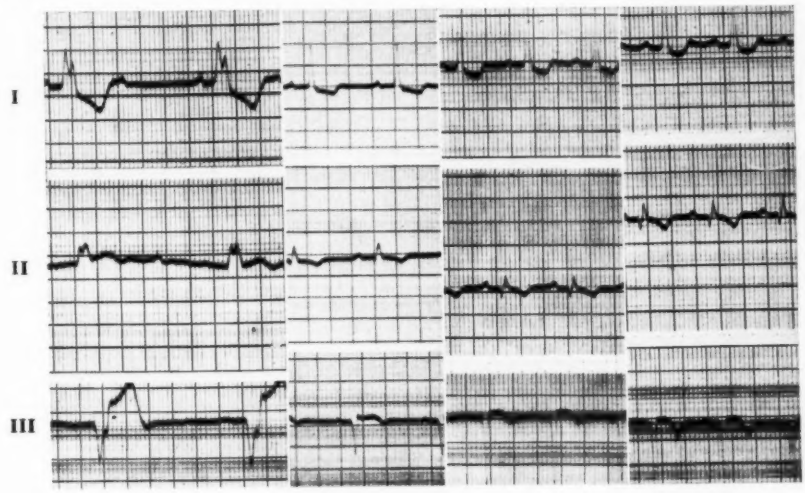

Fig. 2. Electrocardiographic progress studies in a subject with infarction of the posterior wall of the left ventricle. Intervals after coronary occlusion: A—6 days; B—12 days; C—54 days; D—68 days.

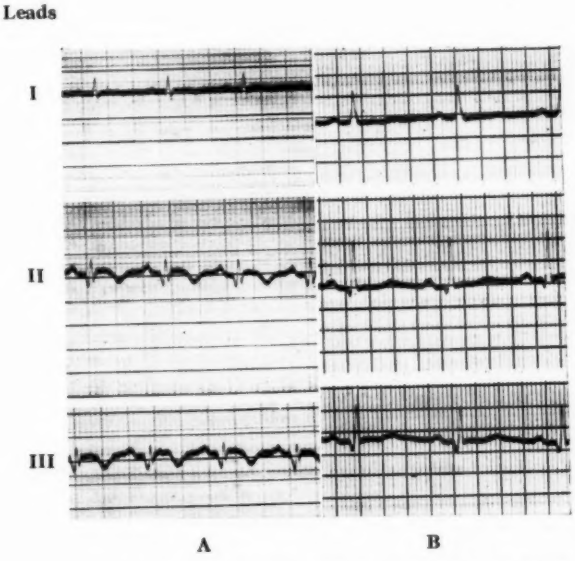

Fig. 3. Marked electrocardiographic recovery after infarction of the posterior wall of the left ventricle. Intervals after coronary occlusion: A—21 days; B—4.5 years.

farcts, extensive overriding infarcts or in infarction complicated by pericarditis.

In few clinical problems does the outcome depend so completely upon the absolute coöperation of the patient as does coronary occlusion. Rest is imperative and during the acute phase of the episode, this implies release from mental as well as physical stress and strain. Complete rest in bed must be enjoined for a period of at least six weeks, or longer if the clinical indications persist. The recurrence of paroxysmal dyspnea, cardiac consciousness, fatigue or other suggestive details should be taken as indications for caution and extended rest. The use of sedative drugs begins with the frank inception of the occlusion. Here morphine is of great value in quieting the patient, although it is usually ineffectual in controlling the pain. Morphine must be used in full therapeutic doses. There is no standard or mean. After the acute attack particular precautions should be taken to limit the patient's contacts and to exclude from the sickroom the cares and excitements of the daily life of the household, business and community. Visitors should be reduced to a number and type that will not tax the strength of the patient nor exact needless expenditure of energy. The patient must not be expected to entertain. Mildly sedative drugs as the bromides and the barbiturates may serve the useful purpose of raising the patient's threshold of reactivity, thereby reducing his response to external stimuli.

Oxygen is beyond a question of doubt the most useful drug at our avail in coronary occlusion. Rizer[72] first suggested its clinical use in this relation in 1929. Using atmospheres containing 50 volumes per cent oxygen he remarked relief from pain in as short an interval as two minutes. This result has been repeatedly confirmed by many clinicians. The reports of Levy and Barach[2,3,47] have been particularly convincing. They state, "In some instances effective use of oxygen may be responsible for the saving of life." These workers utilize a 50 volumes per cent oxygen in a tent for five days. Our experience has led us to use 40 to 60 volumes per cent oxygen through a nasal catheter over a period of at least two weeks. The manner of administration is of secondary importance. A decline in the general condition with an acceleration of the pulse rate attends premature withdrawal of this support. As measures of its advantage Barach and Levy[3] cited the following: striking subjective improvement in the relief of pain, less labored and slower respiration, control of the restlessness (sufficient to eliminate the need of morphine), diminution or abolition of the cyanosis and Cheyne-Stokes respiration (if present), fall in the temperature (if elevated), slowing of the heart rate and improvement in the general circulatory state.

The mechanism of these remarkable results from oxygen therapy in coronary occlusion are not entirely self-evident. Of course, it is clear that the anoxemia resultant upon the embarrassed circulation is relieved and the functional capacity of the myocardium is forthwith improved. This circumstance might explain all of the benefits from oxygen inhalation in coronary occlusion with the conspicuous exception of the relief of the excruciating pain. Haldane[28] has written, "Partial anoxemia means not a mere slowing down of life, but progressive and perhaps irreparable damage to living structure." MacWilliam and Webster[53] in a series of ergometer experiments upon the "congested" and "ischemic" arm found that there was little sensation with the arm at rest but on effort the fatigue point was speeded and the fatigue was acutely painful. This result was likened to angina where pain occurred upon effort in the presence of an inadequate blood supply to the myocardium. Lewis,[48] Resnik[71] and others have demonstrated the apparent causal relationship of anoxemia and the products of asphyxia to a series of faults in conduction. In 1931 Lewis, Pickering and Rothschild[49] convincingly proved that there occurred no vascular spasm concomitant with the pain of effort with inadequate blood supply. They postulate the existence of a P factor, a metabolite developed within the muscle but not directly resultant from the anoxemia. Its accumulation and the excitation of pain arise from faulty circulatory removal. Kissin[41] noted that pain in the exercised muscles of the forearm after arterial constriction was relieved by the inhalation of oxygen. The electrocardiographic curves of coronary occlusion have been closely approximated by local and general anoxemia.[20,40,42,76]

From a clinico-pathologic viewpoint it has been difficult to reconcile the relief of pain from oxygen therapy in coronary occlusion to a simple control of anoxemia. The zone of myomalacia

is strictly speaking without the free circulation. Hence its oxygenation can not be favored by raising the available oxygen in the respired air. The myocardial nutrition is favored by this expedient and it is conceivable that the mobilization of the P factor (or other metabolite) as suggested by Rothschild and Kissin[73] and by Katz[39] may be facilitated by this procedure. A further insight into the biochemical reactions attendant upon coronary occlusion is afforded by the work of Himrich, Goldfarb and Nahum.[34] Before the ligation of the coronary artery the heart removed lactic acid from the blood, whereas after such ligation lactic acid was added to the blood by the heart. The diminished oxygen supply to the myocardium may account for this increase in lactic acid production and conversely its increase may limit the abnormal metabolism. For the present purpose it is important to bear in mind that oxygen inhalations are effective in relieving the pain of coronary thrombosis in a high percentage of cases and coincidentally other significant advantages are gained.

As the logical and sustained source of energy the diet requires especial attention in myocardial affections. Since glucose is the most readily combustible of foodstuffs, it should be especially maintained in the dietary of such patients. From the precepts of Budingen[9] has arisen a tendency to feed excesses of glucose to patients with myocardial disorders. Allan,[1] for example, advised one-half pound of glucose daily in coronary thrombosis. Master, Jaffe and Dack[54] sounded a reactionary note in advising low caloric diets under these conditions. On an 800 calorie diet (80 gm. carbohydrate, 50 gm. protein and 30 gm. fat) declines of the basal metabolic rate to —20 and —30 per cent were reported. Apparently incidental and more serious hazards have been overlooked by these workers in their desire to attain a fixed end. Experimental proof of the efficacy of glucose is available in the observations of Edmunds and Cooper[17] of the life-saving property of the intravenous injection of 10 per cent glucose solution in the circulatory failure incident to intoxication by diphtheria toxin. The excellent clinical results of Smith, Gibson and Ross[81] from the use of high carbohydrate diets in cardiac failure afford adequate argument in support of this plan of procedure. If the stomach be retentive, it is obviously unnecessary to consider other routes of administration; but where nausea and vomiting preclude the alimentary route, we have for years been giving a 5 per cent glucose solution intravenously in 500 c.c. doses two or three times a day. Admittedly this low concentration of glucose affords a relatively small amount of energy; but its isotonicity obviates the serious danger of the right heart overload that sometimes attends the use of hypertonic glucose solution.

After the acute danger is past the diet should still be arranged to meet the energy requirements of a laboring myocardium. Certainly a balanced ration with adequate carbohydrate is in order. The fluid intake should not exceed 1,500 c.c. and the salt should not be pushed. In fact it is safer to maintain a salt-poor regimen, if there be signs of congestive failure. Overloading of the stomach must be assiduously avoided in view of the symptoms thereto attendant in coronary disease. Five small rather than three average meals should be the rule. Abdominal distension must be avoided or corrected because of the respiratory and circulatory handicaps it imposes. For the first few days little attention need be paid to the state of the bowels. Thereafter it is well to insure a loose or slightly formed stool by the use of mild cathartics. Non-irritant enemata may effect the same end. The serious burden of straining at stool[55] should never be overlooked in this group of patients.

Efforts to improve the blood flow through the coronary system have been rewarded by both experimental and clinical encouragement. In 1925 Smith, Miller and Graber[82] in an experimental study upon rabbits found that perfusions of 1-25,000 caffein sodio-benzoate solution and of 1-25,000 theobromine sodio-salicylate did not influence the cardiac rate, amplitude of contraction or coronary flow. A strength of 1-12,500 of the latter drug reduced the rate of flow with equivocal results upon the cardiac rate and amplitude. Theophyllin and euphyllin in strengths of 1-25,000 increased the coronary flow independent of increase in the rate. Musser[60] reported results from theophyllin ethylenediamin in the after-care of coronary thrombosis only slightly less favorable than those remarked in angina pectoris. Finally the recent experimental observations of Fowler, Hurevitz and Smith[21] placed the peculiar utility of theophyllin ethylenediamin on a sound basis. Conclusive evidence of its action in promoting collateral circulation

was adduced from the marked restriction of the area of infarction and scarring in dogs receiving the drug after ligation of the coronary artery as compared with the extent of infarction in control animals that received none. Accordingly theophyllin ethylenediamin should be given immediately after the coronary accident, and if necessary it may be administered intravenously. At the Wisconsin General Hospital it has been our plan to continue its use for four to six months. The dose usually advised is 1½ grains (0.1 gm.) three times a day. Occasionally it has been necessary to interrupt the course of the drug by reason of restlessness, insomnia, or other evidence of cerebral stimulation. On the other hand, a patient of mine has taken 1½ grains twice a day for over two years without a recurrence of his symptoms and without ill effects. It has been our custom to discontinue the theophyllin ethylenediamin for trial periods of rest. A return of adverse symptoms is adequate warning for its renewal.

The immediate support of the heart during and succeeding the accident of coronary occlusion next requires our attention. This demand is much less pressing than was the case before the routine use of oxygen, glucose and theophyllin ethylenediamin. Occasionally the circulatory collapse of the initial period requires heroic measures. If there be venous hypertension with dyspnea, cyanosis, pulmonary edema, venous engorgement and enlargement of the liver, a venesection of 500 c.c. may be life-saving. One patient in our series lived for two years after such an experience. Routine observations of the venous pressure are recommended in this relation and a maintained or rising pressure of over 20 cm. of water in the veins may be taken as an absolute indication for phlebotomy.[19] The rapidly acting stimulants, as caffein sodium benzoate, strophanthin and epinephrin, should find an occasional place in this emergency, but they have commonly failed us. By reason of their remarkable efficacy in angina pectoris, it is perhaps natural that the profession should turn and cling to the use of nitrites in coronary thrombosis. Prodger and Ayman[69] have pointed out the potential danger of nitroglycerin in further lowering the blood pressure and thus favoring coronary thrombosis.

Levine[46] has called attention to the serious threat of ventricular tachycardia in coronary thrombosis. For its control he suggested the availability of quinidine, which was first used by Scott[74] for this condition. Levine[46] has used as much as 1.5 gm. of quinidine sulphate five times a day with success. By reason of its evanescent action doses of 2 grains (0.125 gm.) are recommended at 2 hour intervals through the day and night for thirty-six hours. The individual dose may be increased to 4 grains (0.250 gm.); but a longer interval than two hours will not afford a fair trial of the drug's usefulness. Objection is registered against the routine use of depressant agents such as quinidine in coronary occlusion for prophylaxis against so unusual a complication as ventricular tachycardia. Epinephrine has a clear place in the treatment of heart block and it may operate dramatically at times. For more continued use ephedrine sulphate (0.025 to 0.050 gm. three times a day) or barium chloride (0.030 to 0.120 gm. four times a day) may be useful in the abolition of the episodes of Adams-Stokes' syndrome.

The use of digitalis in coronary thrombosis has been the source of considerable controversy. Gold[25] concluded from animal experimentation that ventricular fibrillation, the most serious hazard of digitalis, occurred no more frequently after coronary obstruction than in control animals. Scarcely was this fear allayed when Gilbert and Fenn[24] found that digitalis decreases the coronary flow in dogs by vaso-constriction. In all probability we should withhold digitalis in coronary occlusion unless there be evidences of decompensation or threatened decompensation. It must not be given routinely.

The antisyphilitic measures of therapy should be deferred, where this etiology exists, for six weeks or two months after a coronary accident to assure adequate repair. Then mercury or bismuth and potassium iodide rather than the arsenicals are employed over a period of six weeks to two months. Thereafter arsenicals may be given cautiously and periods of mixed treatment may be interspersed with the arsenical series.

The diabetic subject with coronary thrombosis presents an interesting problem. Earlier occasion[57] has been taken to point out the dangers of insulin shock and hypoglycemia. Budingen[9] has emphasized the clinical hazards of these circumstances. Turner[56] and others remarked the development of anginal attacks incident to in-

sulin shock. Apparently the profession has taken a most reactionary viewpoint in the matter. There can be no doubt of the need of insulin in such patients and the real precaution that must never be overlooked is the avoidance of insulin shock at all costs.

With the reawakened interest in life will come requests from the patient to resume certain habits. It is interesting that recent observations should have cleared coffee and alcohol of any direct responsibility in coronary disease. There is no apparent reason to interdict the use of such beverages in moderation. Tobacco has a different charge. Lampson[43] has very recently completed a very interesting and convincing study of the vaso-constricting effect of smoking. After smoking and inhaling a cigarette marked vasoconstriction lasting for about 60 minutes was observed in the peripheral vessels. If the subject did not inhale, this vasoconstriction lasted only 15 minutes. Denicotinized cigarettes had less effect than untreated cigarettes. The cigar and pipe induced parallel effects to the cigarette. Hence tobacco in all forms must be forbidden in the patient who has sustained a coronary occlusion.

Finally the patient himself must be a full partner in the conduct of his later convalescent course and in his readjustment to a more or less modified plan of subsequent living. It is rarely wise to take the patient into your complete confidence in the shocking hours or days immediately succeeding the coronary accident. The immediate family or friends will share the confidence of the physician at this time. As the patient begins to extend his interests beyond the sickroom, it is wise to give him some appreciation of the gravity of the situation. The approach must be most tactfully made and only enough of the truth told to insure his complete coöperation. The problem is strictly individual and the physician's insight into the psychology of the patient will ease the path and avoid needless shock. It is well to give him an approximate idea of the protracted period of inactivity that faces him relatively early in the convalescence. The degree of ultimate recovery depends so much upon the completeness of his coöperation that the very idea of a lack of definition of his program may well wreak havoc. The transition to ordinary daily life and activities must of necessity be very gradual in an effort to avoid the retracing of steps. There is no royal road to estimating the degree of functional recovery in the myocardium. It must be a program of trial and error. In certain instances the circulatory capacity may regain its normal level. In others it may strike a plateau far below its preocclusive state. To this group, we would direct your attention in closing. Theoretically (or should we say from a functional standpoint?) life may be extended by a sedentary existence and the avoidance of every semblance of strain or effort. Perhaps by these most rigid measures of protection the individual may eke out a few more months or years than would otherwise be the case. On the other hand, he may by following his natural inclination shorten his life by the same measure. In my judgment he has a perfect right to that decision. "Why ward off death if in the attempt we kill living?"[51] A respected physician of Wisconsin came into my office a little while past for an evaluation of his circulatory reserve. When appraised of the existence of a coronary thrombosis, he remarked that he had suspected the same; but said he, "I have no fear of death, just so the twilight is not too long."

References

1. Allan, G. A.: Diseases of the coronary arteries. Brit. M. J., 2:232, 1928.
2. Barach, A. L.: The therapeutic use of oxygen in heart disease. Ann. Int. Med., 5:428, 1931.
3. Barach, A. L., and Levy, R. L.: Oxygen in the treatment of acute coronary occlusion. Jour. A. M. A., 103:1690, 1934.
4. Barnes, A. R.: Electrocardiogram in myocardial infarction. Arch. Int. Med., 55:457, 1935.
5. Barnes, A. R., and Ball, R. G.: The incidence and situation of myocardial infarction in one thousand consecutive postmortem examinations. Am. J. Med. Sc., 183:215, 1932.
6. Barnes, A. R., and Whitten, M. B.: Study of the R-T interval in myocardial infarction. Am. Heart J., 5:142, 1929.
7. Bellet, S., Gouley, B. A., and McMillan, T. M.: The nourishment of the myocardium through the Thebesian vessels in a heart in which the large coronary arteries and veins were destroyed by the tuberculous myocarditis. Arch. Int. Med., 51:112, 1933.
8. Bickel, G., Mozer, J., and Sciclounof, F.: La sédimentation globulaire dens l'infarctus du myocarde. La signification diagnostique et pronostique. Arch. des Malad. du Coeur, des Vaisseaux et du Sang, 28:73, 1935.
9. Budingen, T.: Ernährungs-und Stoffwechsel-Störungen in Herzen (Kardiodystrophien) beim Di-

abetes Mellitus und beim "Insulinschaden." Zentralbl. f. Herz. u. Gefassk., 17:215, 231, 1925.
10. Burak, M.: Die Bedeutung der Senkungsgeschwindigkeit für die Diagnose des akuten Coronarverschluss. Wien. klin. Wchnschr., 47:327, 1934.
11. Burton, J. A. G., Cowan, J., Kay, J. H., Marshall, A. J., Rennie, J. K., Ramage, J. H., and Teacher, J. H.: Four cases of fibrosis of the myocardium with electrocardiographic and postmortem examinations. Quart. J. Med., 23:293, 1930.
12. Christian, H. A.: Cardiac infarction (coronary thrombosis); an easily diagnosable condition. Am. Heart J., 1:129, 1925.
13. Conner, L. A., and Holt, E.: The subsequent course and prognosis in coronary thrombosis. Am. Heart J., 5:705, 1930.
14. Cooksey, W. B.: Coronary thrombosis follow-up studies with especial reference to prognosis. Jour. A. M. A., 104:2063, 1935.
15. Diamond, M.: Calcification of myocardium in a premature infant. Arch. Path., 14:137, 1932.
16. Dock, G.: Notes on the coronary arteries, Ann Arbor, 1896.
17. Edmunds, C. W., and Cooper, R. G.: Action of cardiac stimulants in circulatory failure due to diphtheria. Jour. A. M. A., 85:1798, 1925.
18. Evans, J. A.: Coronary occlusion with survival. Minn. Med., 13:163, 1930.
19. Eyster, J. A. E., and Middleton, W. S.: Venous pressure as a guide to venesection in congestive heart failure. Am. J. Med. Sc., 174:486, 1927.
20. Feil, H. S., Katz, L. N., Moore, R. A., and Scott, R. W.: The electrocardiographic changes in myocardial ischemia. I. The effects of ligation of the left descending coronary artery with and without occlusion of the inferior vena cava. Am. Heart J., 6:522, 1931.
21. Fowler, W. M., Hurevitz, H. M., and Smith, F. M.: The effect of theophyllin ethylenediamine on experimentally induced cardiac infarction in the dog. Proc. 26th Ann. Meeting, Am. Soc. Clin. Investig., J. Clin. Investig., 13:694, 1934.
22. Fulton, J. T.: Remarks upon the manner of death in coronary thrombosis. Am. Heart J., 1:138, 1925.
23. Galli, G.: Ueber anastomotische Zirkulation des Herzens. Münch. med. Wchnschr., 50, 2:1146, 1903.
24. Gilbert, N. C., and Fenn, G. K.: Effect of digitalis on the coronary flow. Arch. Int. Med., 50: 668, 1932.
25. Gold, H.: Action of digitalis in the presence of coronary obstruction. Arch. Int. Med., 35:482, 1925.
26. Grant, R. T., and Viko, L. E.: Observations on the anatomy of the Thebesian vessels of the heart. Heart, 15:103, 1929.
27. Gross, L.: The blood supply to the heart, New York, 1921.
28. Haldane, J. S.: Symptoms, causes and prevention of anoxemia, and the value of oxygen in its treatment. Lancet, 2:65, 1919.
29. Hamburger, W. W.: Disease of the coronary vessels, angina pectoris, and "acute indigestion." Med. Clin. N. Amer., 9:1261, 1926.
30. Hammer, A.: Ein Fall von thrombotischem Verschlusse einer der Kranzarterien des Herzens. Wien. Med. Wchnschr., 28:97, 1878.
31. Herrick, J. B.: Clinical features of sudden obstruction of the coronary arteries. Jour. A. M. A., 59:2015, 1912.
32. Herrick, J. B.: Acute obstruction of the coronary artery, Northwest Med., 24:593, 1925.
33. Herrick, J. B.: Some unsolved problems connected with acute obstruction of the coronary artery. Am. Heart J., 4:633, 1929.
34. Himrich, H. E., Goldfarb, W., and Nahum, L. H.: Changes of the carbohydrate metabolism of the heart following coronary occlusion. Am. J. Physiol., 109:403, 1934.
35. Hirsch, C., and Spalteholz, W.: Coronararterien and Herzmuskel (Anatomische und experimentelle Untersuchungen). Deutsch. Med. Wchnschr., 20: 790, 1907.
36. Hirschboeck, F. J.: Calcification of the myocardium following coronary occlusion. Am. Heart J., 10:264, 1934.
37. Hudson, C. L., Moritz, A. R., and Wearn, J. T.: The extracardiac anastomoses of the coronary arteries. J. Exp. Med., 56:919, 1932.
38. Jegorow, B.: Die intravitale Diagnose des Myokard-infarktes. Ztschr. f. klin. Med., 106:71, 1927.
39. Katz, L. N.: Mechanism of pain production in angina pectoris. Am. Heart J., 10:322, 1935.
40. Katz, L. N., Feil, H. S., and Scott, R. W.: The electrocardiogram in pericardial effusion. II—Experimental. Am. Heart J., 5:77, 1929.
41. Kissin, M.: Relation of induced anoxemia to the pain of muscular exercise. Proc. Soc. Exp. Biol. and Med., 30:114, 1932.
42. Kountz, W. B., and Gruber, C. M.: Electrocardiographic changes in anoxemia of the heart. Proc. Central Soc. Clin. Research, Nov. 22, 1929, J. Clin. Investig., 8:664, 1930.
43. Lampson, R. S.: A quantitative study of vasoconstriction induced by smoking. Jour. A. M. A., 104: 1963, 1935.
44. Leary, T., and Wearn, J. T.: Two cases of complete occlusion of both coronary orifices. Am. Heart J., 5:412, 1930.
45. Levine, S. A.: Cases of coronary occlusion with recovery. Med. Clin. N. Amer., 8:1719, 1925.
46. Levine, S. A.: Coronary thrombosis, its various clinical features. Baltimore, 1929.
47. Levy, R. L., and Barach, A. L.: The therapeutic use of oxygen in coronary thrombosis. Jour. A. M. A., 94:1363, 1930.
48. Lewis, T.: Observations upon disorders of the heart's action. Heart, 3:279, 1911-1912.
49. Lewis, T., Pickering, G. W., and Rothschild, P.: Observations upon muscular pain in intermittent claudication. Heart, 15:359, 1931.
50. Libman, E.: The importance of blood examination in the recognition of thrombosis of the coronary arteries and its sequelæ. Am. Heart Assoc. Trans., May 26, 1925; Am. Heart J., 1:121, 1925.
51. Longcope, W. T.: Methods and medicine. Bull. J. Hopkins Hosp., Jan., 1932.

52. Luten, D.: Coronary thrombosis of lesser severity. Med. Clin. N. Amer., 11:445, 1927.
53. MacWilliam, J. A., and Webster, W. J.: Sensory phenomena associated with defective blood supply to working muscles. Brit. M. J., 1:51, 1923.
54. Master, A. M., Jaffe, H. L., and Dack, S.: Low basal metabolic rates obtained by low calorie diet in coronary artery disease. Proc. Soc. Exp. Biol., and Med., 32:779, 1935.
55. Meyer, O. O., and Middleton, W. S.: The influence of respiration on venous pressure. J. Clin. Investig., 8:1, 1929.
56. Middleton, W. S.: Coronary Disease. Minn. Med., 10:743, 1927.
57. Middleton, W. S., and Oatway, W. H., Jr.: Insulin shock and the myocardium. Am. J. Med. Sc., 181:39, 1931.
58. Miller, J. L., and Matthews, S. A.: Effect on the heart of experimental obstruction of the left coronary artery. Arch. Int. Med., 3:476, 1909.
59. Moritz, A. R., Hudson, C. L., and Orgain, E. S.: Augmentation of the extracardiac anastomoses of the coronary arteries through pericardial adhesions. J. Exp. Med., 56:927, 1932.
60. Musser, J. H.: Theophyllin ethylenediamin in heart disease associated with pain. Jour. A. M. A., 91:1242, 1928.
61. Oberhelman, H. A., and LeCount, E. R.: Variations in the anastomoses of the coronary arteries and their sequences. Jour. A. M. A., 82:1321, 1924.
62. Obratzow, W. P., and Strachesko, N. C.: Zur Kenntnis der Thrombose der Koronarterien des Herzens. Ztschr. f. klin. Med., 71:116, 1910.
63. Oppenheimer, B. J., and Rothschild, M. A.: The value of the electrocardiogram in the diagnosis and prognosis of myocardial disease. Trans. Assoc. Am. Phys., 39:247, 1924.
64. Pardee, H. E. B.: Heart disease and abnormal electrocardiograms. Am. J. Med. Sc., 169:270, 1925.
65. Pardee, H. E. B.: Complete clinical recovery after thrombosis of a coronary branch. Am. Heart J., 2:442, 1927.
66. Parkinson, J., and Bedford, D. E.: Cardiac infarction and coronary thrombosis. Lancet, 1:4, 1928.
67. Parkinson, J., and Bedford, D. E.: Successive changes in the electro-cardiogram after cardiac infarction (coronary thrombosis). Heart, 14:195, 1927.
68. Porter, W. T.: Further researches on the closure of the coronary arteries. J. Exp. Med., 1:46, 1896.
69. Prodger, S. H., and Ayman, D.: Some harmful effects of nitroglycerine, especially in coronary thrombosis. Am. J. Med. Sc., 184:480, 1932.
70. Rabinowitz, M. A., Shookhoff, C., and Douglas, A. H.: The red cell sedimentation time in coronary occlusion. Am. Heart J., 7:52, 1934.
71. Resnik, W. H.: Observations on the effect of anoxemia on the heart. I—Auricular-ventricular conduction; II—Intraventricular conduction; III—Changes in the auricles, with particular reference to the relationship between anoxemia and auricular fibrillation. J. Clin. Investig., 2:93, 117, 125, 1925.
72. Rizer, R. I.: Oxygen in the treatment of coronary occlusion. Minn. Med., 12:506, 1929.
73. Rothschild, M. A., and Kissin, M.: Anginal syndrome induced by gradual general anoxemia. Proc. Soc. Exp. Biol. and Med., 29:577, 1932.
74. Scott, R. W.: Observations on a case of ventricular tachycardia with retrograde conduction. Heart, 9:297, 1921.
75. Scott, R. W.: Syphilitic aortic insufficiency. Arch. Int. Med., 34:645, 1924.
76. Scott, R. W., and Katz, L. N.: The electrocardiogram in pericardial effusion. I—Clinical. Am. Heart J., 5:68, 1929.
77. Singer, R.: Zur Klinik der Coronar-Thrombose. Wien. klin. Wchnschr., 26:810, 1934.
78. Slater, S. R., and Kornblum, D.: Bilateral coronary occlusion with mitral stenosis. Jour. A. M. A., 102:38, 1934.
79. Smith, F. M.: The ligation of coronary arteries with electrocardiographic study. Arch. Int. Med., 22:8, 1918.
80. Smith, F. M.: Further observations on the T wave of the electrocardiogram of the dog following the ligation of the coronary arteries. Arch. Int. Med., 25:673, 1920.
81. Smith, F. M., Gibson, R. B., and Ross, N. G.: The diet in the treatment of cardiac failure. Jour. A. M. A., 88:1943, 1927.
82. Smith, F. M., Miller, G. H., and Graber, V. C.: The effect of caffein sodio-benzoate, theobromin socio-salicylate, theophyllin and euphyllin on the coronary flow and cardiac action of the rabbit. J. Clin. Investig., 2:157, 1925.
83. Spalteholz, W.: Die Arterien der Herzwand. Leipzig, 1924.
84. Stella, G.: The part played by the Thebesian vessels in the blood supply to the heart. J. Physiol., 73:36, 1931.
85. Thorel, C.: Pathologie des Kreislauforgane. Ergebn. allg. Path. u. path. anat., 9, pt. 1; 673, 1903.
86. Turner, K. B.: Insulin shock as a cause of cardiac pain. Am. Heart J., 5:671, 1930.
87. von Leyden, E.: Ueber die Sclerose der Coronar-Arterien and die davon abhangigen Krankheitszustande. Ztschr. f. klin. Med., 7:459, 1884.
88. Wayne, E. J., and Graybill, A.: Observations on the effect of food, gastric distension, external temperature, and repeated exercise on angina of effort, with a note on angina sine dolore. Clin. Science, 1:287, 1934.
89. Wearn, J. T.: Role of the Thebesian vessels in the circulation of the heart. J. Exp. Med., 47:293, 1928.
90. Wearn, J. T., Mettier, S. R., Klumpp, T. G., and Zschiesche, L. J.: The nature of the vascular communications between the coronary arteries and the chambers of the heart. Am. Heart J., 9:143, 1933.
91. Weigert, C.: Ueber die pathologischen Gerinnungsvorgänge. Arch. f. Path. Anat. u. Phys. u. f. klin. Med., 79:87, 1880.
92. White, P. D.: The prognosis of angina pectoris

and of coronary thrombosis. Jour. A. M. A., 87: 1525, 1926.
93. White, P. D.: Longevity after coronary thrombosis. Jour. A. M. A., 100:233, 1933.
94. White, P. D., and Bland, E. F.: A further report on the prognosis of angina pectoris and of coronary thrombosis. Am. Heart J., 7:1, 1931.
95. Willius, F. A.: Prognosis in heart disease with special reference to coronary disease. Minn. Med., 10:267, 1927.
96. Wollheim, E.: Herzinfarkt und Angina pectoris. Deutsch. Med. Wchnschr., 57:617, 1931.

CORRELATION OF X-RAY HEART SHADOWS WITH CLINICAL AND AUTOPSY FINDINGS*

ALFRED HOFF, M.D.
Saint Paul

THE roentgen ray study of the heart and great vessels has been advanced by a large number of workers, among whom should especially be mentioned Vaquez and Bordet, Herbert Assmann, and F. M. Groedel.

Much of this work has been an effort to demonstrate the changes in the cardiac outline due to disease or congenital defects, and to establish exact methods of measurement.

The size and shape of the normal heart is most difficult to determine because it is affected by height, weight, race, age, the position and contour of the diaphragm and structural deformities of the thorax. In addition, the roentgen rays being divergent rays, distortion of the cardiac silhouette results unless the subject is placed at a seven foot film distance or use is made of the central ray as in orthodiagraphy. Each of these methods gives a fairly accurate estimate of heart size which may be expressed in a percentage value, known as the cardio-thoracic index, on the basis of the ratio of the total transverse diameter of the heart to that of the thorax at its widest inside diameter, usually at the costo-diaphragmatic attachments.

Although the cardio-thoracic index correlates the body type and the heart size in an inaccurate manner, it has the clinical advantage of speed and of affording the clinician some numerical conception of smallness or bigness. Unless heart measurements take into consideration body types, they have little significance.

The lesser degrees of hypertrophy are most difficult of accurate demonstration. Lewis says that "great accuracy of measurement is unattainable and, if it were, it would serve no practical purpose."

The position of the diaphragm and the constitutional habitus of the individual especially affect the shadow contour of the normal heart. At full sustained inspiration, the heart of the slender subject will give a fairly straight left border, the so-called mitral configuration. At full sustained expiration, that of the short, stout or obese individual will conform to the aortic type in which the usual concavity of the left border becomes markedly accentuated. In the first instance, it is probable that in a long, narrow chested person, the forces acting upon the heart during the descent of the diaphragm rotate it to the left, thereby increasing the normally prominent conus pulmonalis and straightening out the left border. In the short, stout individual, who usually has a high diaphragm, deep expiration has the opposite effect.

The phase of respiration, however, does not appreciably affect the shadow contour of definitely diseased large hearts because with enlargement there is usually an accompanying change of form. This is seen in valvular defects where frequently the cardiac silhouette alone often permits of a diagnosis of the underlying pathological condition.

Although the cardiac silhouette merely reveals evidence of gross structural change, which is usually impossible of correction, it does aid materially in the establishment of exact diagnosis and accuracy of prognosis which is so essential to the intelligent care of cardiac patients. Especially is this true in the older age

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group where auricular fibrillation and congestive heart failure by modifying and obscuring the characteristic murmurs of old valve defects cause them to simulate other types of failure which are more common to that group.

Chronic vavular disease in the young adult or child, where the characteristic murmurs are present, is usually quite easily diagnosed without the assistance of the roentgen ray.

The main purpose of this paper is to illustrate, by the shadow contour of the heart and great vessels alone, the changes occurring as a result of disease and to correlate this picture with the clinical and autopsy findings, where possible, without resorting to fixed cardiac measurements. The cases to be shown consist of those which have been under personal observation for variable periods of time. The films have been taken at medium sustained inspiration in the antero-posterior position at six foot film distance. This permits of easier taken comparable films and of greater film contrast owing to the increased aeration of the lungs.

The left and right anterior oblique positions as a supplement for more detailed information regarding the encroachment of the heart and aorta upon the retrocardial space and mediastinum are very important. However, in practice, these positions are usually done during fluoroscopy, with the result that permanent records for presentation are not available.

The visible contours as shown in the x-ray silhouette of the normal heart and aorta consists on the right side of the outward curve of the right auricle, terminating in that of the ascending aorta, which hardly extends beyond the spinal column. This junction is usually well defined, giving a good estimate of their relative position and length. It is to be noted that the ascending aorta is somewhat longer and in a different vertical plane from that of the right auricle. These differences are markedly changed in certain disease conditions.

The extension of the shadow upwards and to the right is caused by the superior vena cava while that of the aorta is seen to the left as a rather small prominent rounded shadow, called the aortic knob. This represents the beginning of the descending aorta.

The left contour is usually described as consisting of four arcs, the aortic, pulmonary, left auricular and left ventricular. However, general opinion holds that when the pulmonary or left auricular arcs can be definitely identified, the heart is abnormal.

The central part of the heart shadow is produced almost entirely by the right ventricle, with the left ventricle occupying the lower left lateral margin.

In compensated mitral stenosis, there is a definite posterior enlargement of the left auricle. The resultant increased pressure in the lesser circulation causes hypertrophy of the right ventricle. As it enlarges it pushes the right auricle outwardly, making prominent and higher the junction of the right auricle with the ascending aorta. Its enlargement also causes the heart to rotate to the left on its vertical axis, thereby making the conus pulmonalis prominent, straightening out the left border and obscuring the aortic knob. The heart shadow becomes ovoid in form and shows little enlargement. In one-half the cases, the left ventricle participates in this enlargement and the extreme lower left contour, therefore, becomes modified by the amount of the left hypertrophy (Plate 2).

As mitral insufficiency is added, the left ventricle elongates, increasing the extent and straightness of the left border and giving a corresponding increase in the size of the silhouette. The hilus shadows are distinctly widened, the lung field markings increased in intensity and the costo-diaphragmatic angles indistinct (Plates 3 and 4).

Plate 4 is that of a man, aged twenty-three, who had been treated at a sanatorium for pulmonary tuberculosis, where he had been sent on account of cough with blood streaked sputum. The heart signs were typical of mitral stenosis. The shadow contour reveals the extended straight left border with the absence of the aortic knob, the high right auricular junction with the ascending aorta and the marked congestion of the hilus and lungs.

At autopsy, the mitral valve was stenosed to a 4 mm. orifice and the lungs were negative for tuberculosis.

A marked enlargement of the pulmonary bow, indicating a dilatation of the pulmonary artery, has been usually regarded as typical of a patent ductus Botalli. It has also been interpreted as being caused by pulmonary stenosis.

Assmann was one of the first to show that, although pulmonary stenosis may produce dilata-

tion of the pulmonary artery, it is usually accompanied by an interventricular septal defect in which the dilatation is then entirely absent and the heart shadow consequently approaches the normal configuration, with the exception that the aortic knob is placed higher and to the right of the spinal column.

However, a patent ductus arteriosus may be present and produce a contour similar to that in Case 3 and show only a slight increase in the pulmonary arc.

Plate 5 is that of a young woman twenty-one years old, who, from her earliest recollection, had shortness of breath on exertion, associated with palpitation of her heart. There was no apparent cyanosis. Over the precordium, a distinct thrill was palpable, the heart was markedly enlarged to the left and a loud systolic followed by a soft diastolic murmur was heard most intensely at the base. The electrocardiogram was of the inverted type with a normal sinus rhythm. The extended straight left border is modified by a slightly increased prominence of the pulmonary bow and by a definite enlargement of the left ventricle.

At autopsy, all the valves were normal and a patent ductus arteriosus was found.

In this instance, the history, clinical signs and the electrocardiogram pointed the way to the correct diagnosis.

The lesser degrees of increased pulmonary bow prominence may also be seen in conditions in which there is a greatly increased pressure in the lesser circulation, but the shadow heart contour does not conform to the types just illustrated nor the clinical picture to recognized types of heart disease.

Marked cyanosis, associated with intractable asthmatic attacks, polycythemia and clubbing of the fingers, were the presenting symptoms of the patient whose heart outline also shows a moderate dilatation of the pulmonary bow without the characteristic mitral stenotic configuration.

This patient, at autopsy, had a marked hypertrophy of his right ventricle, without evidence

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Plate 1.—Shadow heart contour. Normal type.

Plate 2.—Mitral heart contour. Ovoid type. M. S., aged eighteen years. *Clinical diagnosis:* Compensated mitral stenosis. *Clinical findings:* History of rheumatic fever. Sharp first apical tone. *Electrocardiogram:* Partial right axis deviation.

Plate 3.—Mitral heart contour. Mrs. H. S., aged fifty years. *Clinical diagnosis:* Mitral stenosis and insufficiency.

Plate 4.—Mitral heart contour. D. McD., aged twenty-three years. *Clinical diagnosis:* Chronic valvular heart disease. Mitral stenosis and insufficiency. Congestive heart failure with slight edema. *Autopsy findings:* (1) Heart weight 528 gms. (2) Old valve defect (mitral stenosis). Mitral leaflets thickened, stenosed and calcified. Orifice 4 mm. x 1:5 cm. Aortic, pulmonary and tricuspid valves normal. (3) Right ventricular hypertrophy. (4) Myocardinm, pale and swollen. (5) Left pleura adherent to pericardium. (6) Chronic passive congestion of lungs, liver and spleen.

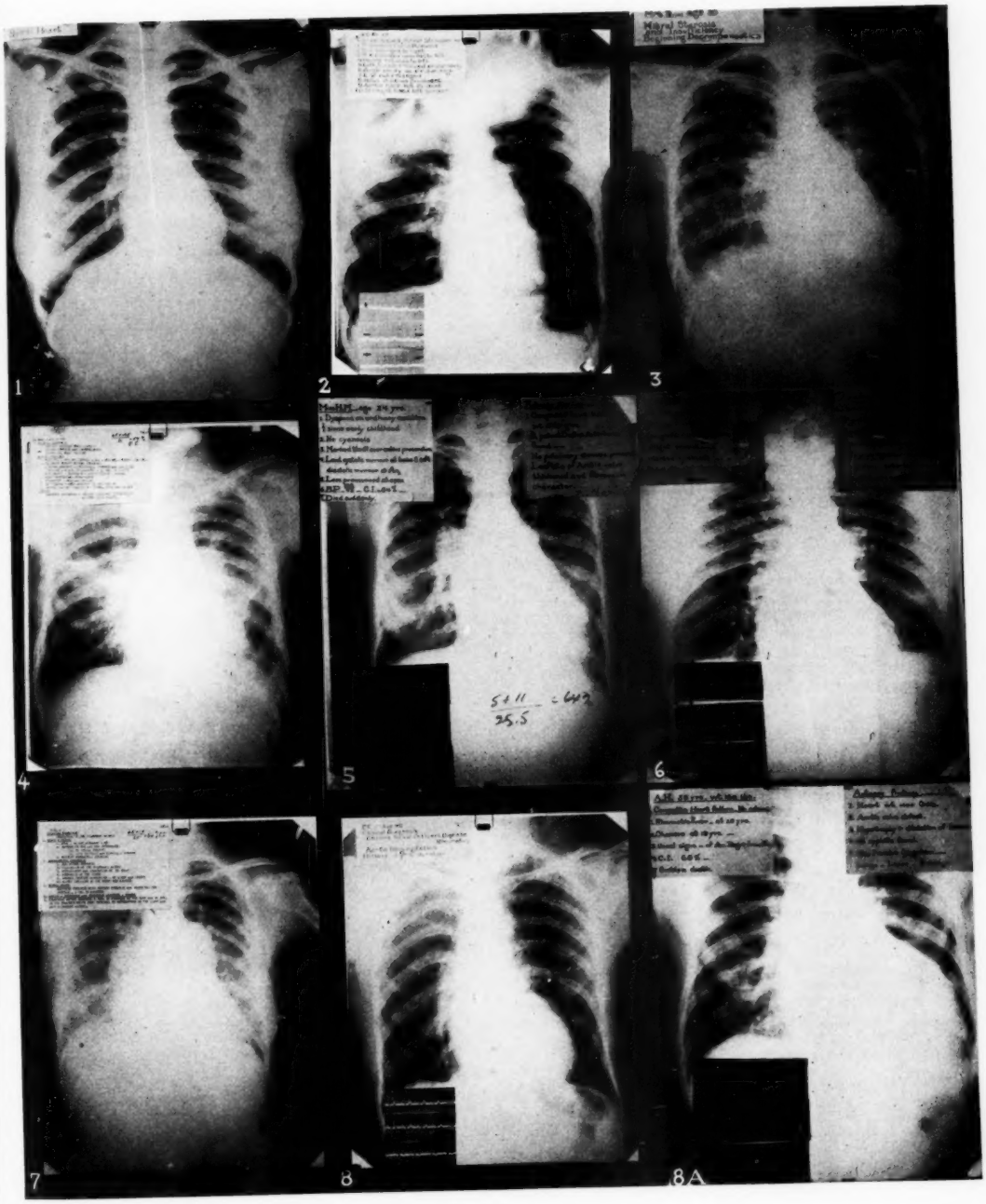
Plate 5.—Mitral heart contour. Miss H. M., aged twenty-four years. *Clinical diagnosis:* Patent ductus arteriosus. Congenital heart disease. *Autopsy findings:* (1) Congenital heart disease. Weight 900 gms. (2) Patent ductus arteriosus, 5 mm. in diameter. (3) No pulmonary stenosis present. (4) Leaflets of aortic valve thickened and fibrous in character.

Plate 6.—Pulmonary arc prominence. Ayerza's disease. W. R., aged forty-three years; weight 170 pounds. *Clinical findings:* Severe dyspnea, cough and asthmatic attacks for five years. Congestive heart failure with slight edema. No murmurs. Marked cyanosis. Polycythemia. Hemoglobin 115; red blood cells, 7.5 mil. *Electrocardiogram:* Right axis deviation. *Autopsy findings:* (1) Heart weight 496 gms. (2) Right heart failure. Definite hypertrophy and marked dilatation of right ventricle. (3) Four leaflets, pulmonary valve. (4) Pulmonary congestion and edema. No pulmonary arteriosclerosis. (5) Atelectasis, right. (6) Bilateral hydrothorax.

Plate 7.—Mrs. H. S., aged fifty years. *Clinical diagnosis:* Acquired dilatation of pulmonary artery. *Heart findings:* Apex to anterior axillary line. Impulse in third and fifth left interspace. No thrill present. Systolic and diastolic murmurs. Moderate cyanosis. *Pathologic diagnosis:* (1) Old mitral stenosis. (2) Dilatation of pulmonary artery. (3) Hypertrophy and dilatation of heart. (4) Cirrhosis of liver. (5) Chronic passive congestion of liver and spleen. (6) Cloudy swelling of heart and kidneys. *Mitral valve:* Definite stenosis with fibrosis and calcification. Orifice, 1 cm. in diameter. Aortic, pulmonary and tricuspid leaflets normal. Pulmonary artery 8 cm. in diameter at its base and 12 cm. in its greatest width just proximal to bifurcation.

Plate 8.—Aortic type heart contour. C. E. K., aged forty years. *Clinical diagnosis:* Chronic valvular heart disease—rheumatic. Aortic regurgitation, compensated. *Electrocardiogram:* Left axis deviation. Normal T waves L1 and L2.

Plate 8A.—Aortic type heart contour. A. H., aged thirty-nine; weight, 150 pounds. *Clinical findings:* Congestive heart failure. No edema. Rheumatic fever at twenty-nine years. Chancre at nineteen years. Usual signs of aortic regurgitation and insufficiency. Cardiac index 69 per cent. Sudden death *Autopsy findings:* (1) Heart weight, 1,000 gms. (2) Aortic valve defect. (3) Hypertrophy and dilatation of heart. (4) No syphilis found. (5) Chronic passive congestion of lungs, liver and kidneys.



PLATES 1-8A
For description see legends on opposite page.

of pulmonary arteriosclerosis or findings usually evident in bronchial asthma (Plate 6).

A marked dilatation of this arc may be acquired and caused by a mitral stenosis. This was the condition found at autopsy in another patient (Plate 7). She was a cyanotic woman, aged fifty, with a heart markedly enlarged to the left, a palpable impulse in the third left interspace, and loud basal systolic and diastolic murmurs, but without a palpable thrill. Auricular fibrillation was present and she showed a congestive heart failure with moderate edema. No polycythemia was present. The mitral valve was calcified and stenosed to a one centimeter diameter, with a marked dilatation of the pulmonary artery and a slight thickening of the pulmonary leaflets.

Aortic configuration results when the left ventricle becomes hypertrophied through increased work by reason of aortic regurgitation or a hypertension.

The enlargement blunts the apex and markedly increases the concavity of the left border but causes little change in the right auricle or the supra-cardiac shadow unless abnormal aortic changes are also present.

In rheumatic or syphilitic aortic regurgitation, the left ventricle becomes greatly enlarged (Plates 8 and 8 *A*).

In the older age group, aortic valve involvement is not infrequently combined with the mitral as a double valve stenosis or with the mitral and tricuspid as a triple valve stenosis and insufficiency.

In combined valvular disease, when it affects the mitral, aortic and tricuspid valves causing triple valve stenosis and insufficiency, the enlarged heart maintains a decidedly mitral stenotic configuration (Plate 9), which persists when an adherent visceral pericarditis is added (Plate 10 and 10 *A*), while if mitral stenosis is combined with aortic stenosis (Plate 11), or with hyper-

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Plate 9.—Mitral heart contour. Triple valve stenosis and insufficiency. Mrs. O., aged thirty-six years. *Clinical diagnosis:* Combined valvular disease. Congestive heart failure with considerable edema. *Autopsy findings:* (1) Heart weight, 445 gms. (2) Mitral leaflets markedly sealed together, thickened and calcified. Orifice, 1 cm. in diameter. (3) Tricuspid leaflets sealed together and slightly thickened. (4) Aortic leaflets sealed together at the commissures and thickened. (5) Pulmonary leaflets normal. (6) Hypertrophy of left ventricle. Slight hypertrophy of right ventricle.

Plate 10.—Mitral type contour. Triple valve stenosis and insufficiency. Mrs. W. H., aged thirty years. *Clinical findings:* Rheumatic fever—four attacks from eleven to twenty-five years. Four years in hospital with congestive heart failure and marked, constantly re-accumulating ascites and moderate edema. Blood pressure, 120/50. Cardiac index, 60 per cent. Palpable precordial systolic thrill. Systolic and diastolic murmurs heard at aortic area and apex. *P. R.* interval, 0.28 seconds.

Plate 10A.—Mitral type contour. Triple valve stenosis and insufficiency. Terminal stage—four years later. Mrs. W. H., aged thirty-four years. *Clinical diagnosis:* Congestive heart failure with marked edema and ascites. Auricular flutter with regular ventricular response. *Autopsy findings:* (1) Mitral, aortic and tricuspid stenosis. (2) Adherent visceral pericardium. (3) Cirrhosis of liver. (4) Hyloserositis of spleen and liver. (5) Ascites.

Plate 11.—Aortic type heart contour. Mrs. S., aged fifty years; weight, 295 pounds. *Clinical findings:* Congestive heart failure with massive edema. No rheumatic history. Blood Wassermann reaction 4 plus. Blood pressure 160/90 to 110/60. Basal metabolic rate plus 19. Cardiac index, 60 per cent. Harsh systolic murmur at aortic area, second tone muffled and indistinct. Harsh systolic murmur at apex. No palpable thrill. Auricular fibrillation. *Autopsy findings:* (1) Heart weight, 740 gms. Body weight, 280 pounds (estimate). (2) Calcified aortic leaflets with definite stenosis of orifice. (3) Mitral valve stenosis. (4) Hypertrophy and dilatation of heart. (5) General anasarca. (6) No syphilis found.

Plate 12.—Aortic type heart contour. Mitral stenosis and hypertension. Mrs. W. J., aged sixty-four years. *Clinical findings:* Congestive heart failure with slight edema. Angina of effort. Rheumatic fever thirty years ago. Auricular fibrillation. No heart murmurs. Blood pressure, 240/130. Cardiac index, 63 per cent. *Autopsy findings:* (1) Heart weight, 606 gms. Body weight, 180 pounds. (2) Mitral stenosis. (3) Coronary arteries patent. (5) Kidneys grossly negative. (5) Cerebral malacia.

Plate 13.—Mitral type heart contour. Mitral stenosis and insufficiency with slight aortic and tricuspid involvement. Kay J., aged twenty-four years: weight, 120 pounds. *Clinical findings:* Dyspnea on moderate exertion. No rheumatic history. Frequent tonsillitis. Blood pressure, 110/80. Congestive heart failure. Palpable thrill with presystolic and systolic murmurs. No diastolic murmurs. Heart to left of anterior axillary line. *Autopsy findings:* (1) Old valve defect. Mitral markedly adherent. Aortic slightly adherent. Tricuspid slightly thickened. Pulmonary normal. (2) Passive congestion of lungs and liver. (3) Edema and ascites.

Plate 14.—Pericarditis with effusion (early). Rev. W., aged thirty-nine years. *Clinical diagnosis:* Pericarditis with effusion (early). *Autopsy findings:* (See plate 15.)

Plate 15.—Pericarditis with effusion. Rev. W., two weeks later. *Clinical diagnosis:* Pericarditis with effusion (early). *Autopsy findings* (six months later): (1) Tuberculous pericarditis. (2) Old tuberculosis of the hilus lymph nodes. (3) Right hydrothorax. (4) Miliary tuberculosis of left lung.

Plate 16.—Aortic type heart contour. F. W. H., aged seventy-five years. *Clinical diagnosis:* Hypertensive heart disease. Coronary sclerosis. Right bundle branch block. No murmurs.

PLATES 9-16
For description see legends on opposite page.

tension (Plate 12), the heart conforms usually to the aortic type. However, a slight aortic stenosis may coexist with a marked mitral stenosis and not influence the marked mitral outline (Plate 13). That is, the preponderant lesion determines the type of contour, but may not affect the axis deviation in the electrocardiogram.

Clinically, pericarditis with effusion of small or large degree is frequently unsuspected and unrecognized. It may also exist without much added discomfort to the patient. However, such is not always the case and upon its prompt recognition and relief will depend the transformation of a patient from the depths of unrelievable misery to a condition of relative comfort.

Plate 14 is that of a man, aged thirty-four, who, two weeks previously, after exposure, was seized with a persistently severe low substernal and epigastric pain associated with a temperature of 100 degrees. He was unable to lie down on account of this increasing the severity of his pain. Besides moderate dyspnea and cyanosis, he showed an impairment of resonance at the right base with moist râles bilaterally, a distinct enlargement of his heart to the left and a full, tense, tender abdomen. During the next two weeks, his respiration became so labored that suffocation seemed imminent. He was unrelieved by morphine or oxygen. The cardiac dullness had increased to the left, auricular fibrillation and evidences of consolidation were present at the left base with moist râles bilaterally (Plate 15).

When the two films are compared, it is obvious that only a pericardial effusion could cause, in two weeks, such a decided change in the cardiac outline. Aspiration of 1,000 c.c. of blood-tinged fluid from his pericardium gave him almost immediate symptomatic relief. Repeated aspirations during the following six months of his life kept him in relative comfort.

Autopsy revealed tuberculous pericarditis.

Hypertensive and arteriosclerotic heart disease are the outstanding types found in middle and later life and are the main contributors to the high mortality from cardiac disease.

The helpfulness of the x-ray in these types is limited to the recognition of bigness or smallness of an aortic shadow contour. Evidence of pulmonary congestion may assist in determining the presence of early congestive heart failure.

A very large aortic type heart, with cardio-

Plate 17.—Arteriosclerotic heart disease. Mr. R., aged sixty-eight years. *Clinical findings:* Coronary sclerosis. Angina pectoris. Arteriosclerotic aorta. Small heart. Sudden death due to coronary occlusion. Autopsy not performed.

Plate 18.—Aortic type shadow heart contour. J. G., aged thirty-four years, weight 250 pounds. *Autopsy findings:* (1) Hypertension heart. Weight, 753 gms. (2) Bilateral congestion of lungs. (3) Acute congestion of liver. (4) Splenomegaly.

Plate 19.—Dilatation of ascending aorta. C. W. C., aged forty-eight years. *Clinical diagnosis:* Syphilitic aortitis. *Clinical findings:* (1) Substernal oppression. (2) Blood Wassermann reaction 4 plus. (3) Systolic murmur at base. (4) No diastolic murmur. (5) Normal electrocardiogram.

Plate 20.—Aneurysm of ascending aorta. L. L. (colored), aged forty-five years. *Clinical findings:* Severe substernal oppression with nocturnal attacks of suffocation. Cough severe, with expectoration. No edema. Dyspnea on moderate exertion. (All six months' duration.) Blood pressure, 179/105. Urine albumin 2 plus, casts. Blood Wassermann reaction 4 plus. No diastolic murmur. P2—Ao2. Soft apical systolic murmur. *Electrocardiogram:* T wave minus in L1 and L2. Termination six months later from congestive heart failure with massive edema. Autopsy findings: (1) Saccular aneurysm, ascending aorta. (2) No valvular involvement. (3) Coronary arteries patent. (4) Hypertrophy and dilatation of heart. (5) Hypertension heart. (6) Arteriosclerosis of kidneys.

Plate 21.—Diffuse dilatation of thoracic aorta, simulating aneurysm. Mrs. D. N. S., aged sixty years. *Clinical findings:* Blood pressure, 180/100. Congestive heart failure with definite edema. Systolic and diastolic murmurs (precordial). Spinal Wassermann reaction, 4 plus. Blood Wassermann reaction negative. Pupils fixed. K. J. absent. Died suddenly, after copious black stools. *Autopsy findings:* Estimated weight, 140 pounds. (1) Senile ectasia of the aorta. (2) Hypertension heart. Weight, 550 gms. (3) Gastric ulcer with hemorrhage.

Plate 22.—Dilatation of ascending aorta. Syphilitic aortitis. L. W. C., aged fifty years. *Clinical findings:* Blood pressure 170/90. No heart murmurs. Distant heart sounds. Moderate enlargement. *Pathologic diagnosis:* (1) Syphilitic aortitis. (2) Cardiac hypertrophy. (3) Coronary orifice stenosis. (4) Left coronary trunk occlusion. (5) Chronic passive congestion of spleen and kidney. (6) Acute passive congestion of liver.

Plate 22A.—Left anterior oblique position. Syphilitic aortitis. Same case as Plate 22.

Plate 23.—Atherosclerotic dilatation of aortic arch. M. J. H., aged sixty-two years (1926). *Clinical findings:* No cardiac symptoms. Blood pressure, 160/100 (1922). Blood Wassermann reaction negative. Blood pressure, 220/120 (1931). Hemiparesis (transient). *Electrocardiogram:* Normal.

Plate 24.—Atherosclerotic dilatation of aortic arch. M. J. H., aged seventy-one years (nine years later—1935). *Clinical findings:* No cardiac symptoms. Blood pressure, 170/110. Blood Wassermann reaction negative. *Electrocardiogram:* Coronary sclerosis.

PLATES 17-24
For description see legends on opposite page.

graphic evidence of definite myocardial damage, may, nevertheless, be functionally adequate to carry on, while a small arteriosclerotic type of heart, following unusual exertion, may undergo sudden and unexpected dissolution.

Four years ago, I saw a man seventy-five years old, who, after a cocktail and a hearty meal, complained of severe epigastric distress, associated with nausea and vomiting. His face was pale and his skin moist and clammy.

His pulse was of low tension, but not fast. On examination, he had a heart markedly enlarged to the left, with good tones, and a blood pressure of 158/90. The heart was boot shaped with a cardio-thoracic index of 60 per cent. His electrocardiogram showed a right bundle branch block. The next morning he felt perfectly well. He is now seventy-nine years old and during the past four years he has never been incapacitated. He has led a very active life for a man of his age, averaging about three nine hole games of golf weekly throughout the year (Plate 16).

A conclusion based upon the x-ray findings and the electrocardiogram, without due regard to the history of lack of pain, and his prompt recovery, would have led one sadly astray and placed unnecessary restrictions upon his activity.

About this time, another man, sixty-eight years old, who had always led a vigorous, active life, exposed himself to an unusual amount of exertion. He was suddenly seized with a severe uninterrupted substernal pain, which extended into his left arm and little finger. He obtained medical relief and upon his return to the city developed angina on effort. Three weeks later, while resting in a hospital and symptom-free, he died, suddenly, in an agonizing attack of substernal pain, the result of a coronary occlusion.

His heart silhouette (Plate 17) shows merely a small heart with a long aorta such as is frequently seen in arteriosclerosis, while in the electrocardiogram taken a month after his first attack of pain, signs of coronary sclerosis were evident.

Sudden death may also occur in a large heart in which the electrocardiogram gives no evidence of myocardial damage, the x-ray only a suggestion of venous congestion and the postmortem examination no convincing explanation.

This was the case in an obese man, thirty-four years old, who entered the hospital on account of paroxysmal attacks of nocturnal dyspnea, with no past history of hypertension or of a heart affliction and who, a short time previously, had passed a medical examination as fit for heavy work.

The film shows an aortic type silhouette of a markedly enlarged heart involving, especially, the left ventricle, with a cardio-thoracic index of 60 per cent, a clear diaphragm, and a very moderate increase in the lung markings, suggestive of a venous congestion (Plate 18).

This man weighed 250 pounds and, as is so often the case in thick chested, huge individuals, where even extensive enlargement of the heart may be overlooked by palpation and percussion, the roentgen ray proved itself to be most helpful.

His electrocardiogram was not significant. His blood pressure was 130/85, basal metabolic rate plus 16 and his blood Wassermann negative. One arterio-venous constriction was seen in the fundus of his left eye. Throughout his hospital stay of three months, he was comfortable, lying flat in bed, without edema or dyspnea at rest, but he had a persistent gallop rhythm at the apex of his heart and a pulsus alternans, both of which told the story of the seriousness of his heart condition, so that his sudden death was not unexpected.

It is obvious that neither heart size nor the findings in the electrocardiogram can measure heart function. This must be determined by the entire clinical picture and interpreted in the light of clinical experience.

The aging process, with its attendant loss of vessel elasticity, affects the aorta by gradually increasing its length and width. Its size, shape and position will be altered, therefore, according to the adaptation needed to adjust itself to the restrictions of its thoracic cage. This adaptation will be easier of accomplishment in the tall, slender individual, resulting in a long ascending aorta without significant widening.

In the short or thick-set person, where what constitutes a normal aortic arch is often difficult of determination, the addition of the factors of compression and torsion must receive consideration in the estimation of the presence of dilatation. Usually, however, when proper emphasis is given to age, body type, clinical symptoms and findings, lues, often unsuspected, is suggested by signs indicating increased width or dilatation of the ascending aorta. In a considerable number

of cases, lues exists without physical signs of aortic insufficiency or significant electrocardiographic abnormalities. A negative blood Wassermann reaction does not remove it as a possible cause.

Juster and Pardee, in a study of fifty cases of luetic aortitis, found the blood Wassermann negative in 14 per cent, while combined tests on the blood and spinal fluid reduced this percentage to 10 per cent of the total cases. Abnormalities of the T wave in the electrocardiogram occurred in 85 per cent of those with aortic insufficiency and in 38 per cent of those without this lesion.

As the ascending aorta widens and progresses to dilatation, a vertical tangential line passing through its right margin approaches a similar one through that of the right auricle, so that it may coincide or even override, thereby disturbing or reversing their usual relationship. As the aorta also lengthens, the point of junction of the ascending aorta and the right auricle becomes gradually lowered and their normal relation lengths is altered, the right contour of the heart shadow becoming dominated by that of the ascending aorta.

The heart shows no appreciable enlargement unless factors increasing the work of the left ventricle, such as aortic insufficiency or hypertension, are also present.

This dilatation is illustrated in Plate 19, which is that of a man forty-five years old, whose symptoms were epigastric distress and regurgitation of mucus. His gastro-intestinal tract was normal but the fluoroscopic screen revealed a diffusely dilated aortic arch, which, in view of his age, suggested syphilis. His heart revealed a soft basal systolic murmur, his blood pressure was low, his blood Wassermann reaction 4 plus and the electrocardiogram was normal. He obtained considerable relief after specific medication and reduction in his physical activities.

In the case of aneurysm, every part of the aortic arch is affected by the increased width (Thoma's law).

The oblique positions are very helpful in the x-ray examination and in the differentiation of aortitis from other mediastinal tumors.

In the case illustrated, the diagnosis of aortic aneurysm was clinically obvious, the x-ray confirmatory, and the autopsy conclusive (Plate 20). Hypertension and arteriosclerosis of the kidneys were the causative factors in the severe grade of cardiac hypertrophy observed.

However, artherosclerotic dilatation of the aortic arch in the presence of hypertension may give an identical picture with that of a luetic aorta, and be of such an extent as to suggest aneurysmal dilatation. Rarely, the associated clinical symptoms and findings are of such a confirmatory character as to make a differential diagnosis impossible.

The sypmptoms, clinical and roentgen ray findings, as indicated in Plate 21, of a colored woman, aged sixty, who died suddenly of a gastric hemorrhage, would surely justify a diagnosis of luetic aortitis, with the possibility of aneurysmal dilatation, yet the postmortem examination revealed a hypertension heart and senile ectasia of the aorta.

The futility of attempting a differential diagnosis in the presence of hypertension between atherosclerotic and luetic aortic dilatation, in fifth or sixth decade, by the roentgen ray contour without giving adequate consideration to the clinical history, findings and progress may be illustrated by the cases shown in Plates 22, 23, and 24.

Plates 22 and 23 represent, respectively, the dilated aortic arch of a luetic aortitis with hypertension in a man fifty years old and that of an atherosclerotic dilatation with hypertension in a man sixty-one years old. There is considerable similarity between the two and a diagnosis from the shadow contour alone is obviously impossible, even though there is a marked difference in the form of the aortic knob and descending aorta. Some writers have stressed this difference as of diagnostic significance, maintaining that a sharply defined rounded contour of the aortic knob is more characteristic of atherosclerosis, while that of one poorly defined, more of syphilis. This is predicated upon the supposed greater calcium content of the former.

The first patient was observed over a period of eight years. His presenting symptoms were those of a moderately severe diabetes mellitus, with a forty pound weight loss, and nocturnal attacks of severe dyspnea. His blood Wassermann reaction was negative and his blood pressure 170/90. His heart was slightly enlarged but no murmurs were heard.

On account of the diffuse dilatation of his

aortic arch and the association of this finding with heart symptoms and a glycosuria, the possibility of syphilis was considered.

A tentative diagnosis of luetic aortitis, supravalvular type, was made and the patient was given specific treatment with the result that, after two months, he returned to work and continued doing so for a period of eight years, entirely sugar-free unless he ate sugar. During this period, there were several interesting developments.

After his first course of specific treatment, his blood Wassermann became positive and remained fixed thereafter.

His wife developed tabes dorsalis, and had a positive blood Wassermann reaction.

After seven years of observation, soft systolic and diastolic murmurs could be heard at the base of his heart. With this change in physical signs, he gradually developed congestive heart failure and died suddenly. An electrocardiogram made late in the course of the disease shows a left axis deviation with inverted T waves in the first and second leads.

At autopsy, the heart weighed 635 grams. A luetic aortitis involving the commissures and obscuring the orifice of the right coronary artery was present. The left coronary trunk showed a marked intimal thickening with a 75 per cent occlusion. There were white patches of fibrosis throughout a moderately hypertrophied left ventricle. Microscopic changes in the pancreas could not be demonstrated.

The second patient with hypertension showing a diffuse aortic dilatation is still alive and well at the age of seventy-one years. He has been under observation for a period of thirteen years and during this time his blood Wassermann reactions have been repeatedly negative, with and without provocative tests, and except for a transient left hemiparetic attack, when his blood pressure was markedly increased, he has never had a confining illness or enjoyed other than good health.

The interval between the film and the attached electrocardiogram is nine years, and the only significant change is that in the electrocardiogram, which now shows evidence of coronary sclerosis (Plates 23, 24).

We are permitted to believe that in the first patient a luetic aortitis was combined with a hypertension heart and coronary sclerosis, on an arterio-sclerotic basis, and was the determining factor in his rather early demise, while, in the latter, the absence of a luetic factor contributed to his greater comfort and longevity.

Warthin, in a comparision of the incidence of coronary sclerosis, myocardial infarction and coronary thrombosis, in 1,174 autopsies of non-syphilitics and 501 syphilitics, came to the conclusion that syphilis is a predisposing and important factor in the production of arterio-sclerosis of the aorta and coronary arteries, but that the syphilitic lesion, itself, rarely produces coronary sclerosis or occlusion.

Clawson and Bell, however, in a detailed pathologic study of twenty-eight cases, selected from a group of one hundred and twenty-six of syphilitic aortitis, said "that nothing was seen in microscopic sections of the coronary arteries that was different from ordinary senile arterio-sclerosis. That a sclerosis of this kind and degree is not infrequent in persons over forty-five years of age, and apparently not a factor of importance in producing heart failure."

Summary

An attempt has been made to correlate, without resorting to set cardiac measurements, the roentgen ray shadow contour of clinically observed large hearts in adults, with the clinical symptoms and autopsy findings, in an effort to appraise its merits in the diagnosis of the cardiac cases selected.

1. A knowledge of the significance of such contours permits the clinician to obtain rapid information with fluoroscope, alone, and, by use of the orthodiagram, a quick permanent record both of the heart shape and size.

2. It is particularly useful in the combined types of valvular lesions, where auricular fibrillation and congestive heart failure have changed the characteristic murmurs and obscured the type of heart failure.

3. The fluoroscopic screen permits of a rapid and accurate method by which a dilatation of the aortic arch or the pulmonary area can be determined.

4. It offers an accurate method for determining changes occurring in the size and shape of the cardiac outline during the progress of disease, being especially useful in pericarditis with effusion.

5. In determining cardiac function, it offers little help.

6. It may be said that the roentgen ray shadow heart contour is a valuable accessory method in the diagnosis and care of heart disease when combined with the clinical symptoms, findings, and the electrocardiogram.

7. In those painful types of heart disease where often all methods of exact diagnosis fail, it is well to remember the advice of the elder Dr. Janeway, who, in discussing angina pectoris, said, "that, when he disregarded the patient's story and relied solely on his own calm judgment, he usually made a mistake."

I wish to acknowledge my indebtedness to Dr. John F. Noble and Dr. Kano Ikeda for the autopsy records of these patients.

References

Assmann, Herbert: Klinische Roentgendiagnostik der inneren Erkrankungen Dritte Auflage Vogel. Leipsig. pp. 31-155.

Brooks, Harlow: Angina pectoris. Harper's Med. Monograph, p. 127, 1929.

Chandlee, Gertrude Jackson, and Holmes, E. Burville: Clinical and roentgen ray findings in the study of the heart and great vessels. Am. Jour. Med. Sci., 178:364-378, 1929.

Clawson, B. J., and Bell, E. T.: The heart in syphilitic aortis. Arch. Path., 4:922-936, 1927.

Cohn, Alfred E.: Aging of the heart muscle, regarded from a general biologic point of view. Am. Jour. Med. Sci., 177:619-638, 1929.

Juster, L. R., and Pardee, H. E. B.: Abnormal electrocardiograms in patients with syphilitic aortitis. Am. Heart Jour., 5:84-92, 1929.

Köhler, Alban: Roentgenology—the borderline of the normal and early pathological in the skiagram. Rendered into English from the fifth German edition by Arthur Turnball. pp. 336-373.

Lewis, Sir Thomas: Diseases of the Heart. New York: Macmillan Co., 1933. p. 116.

Matthes, M.: Textbook of Differential Diagnosis. Authorized translation of the fourth German edition with extensive additions, by I. W. Held and H. M. Gross. Philadelphia: P. Blakiston's Sons and Co. pp. 431-439.

Rösler, Herman (Vienna, Austria): Personal communication.

Rösler, Herman, and White, Paul D.: Unusual variations in the roentgen shadow of the elongated thoracic aorta. Am. Heart Jour., 6:768-777, 1931.

Warthin, Alfred Scott: Syphilis in the etiology of angina pectoris. Am. Heart Jour., 6:163, 1930.

White, Paul D.: Heart Disease. New York: Macmillan, 1931. pp. 143-183.

White, Paul D., and Camp, Paul D.: A comparison of orthodiagraphic and teleroentgenographic measurements of the heart and thorax. Am. Int. Med., 6:469-481, 1932.

VERRUCA SENILIS AND KERATOMA SENILE*

HAMILTON MONTGOMERY, M.D.

Rochester, Minnesota

I WISH to depict the differences between verruca senilis and keratoma senile because, in spite of recent articles in the literature,[1, 2, 3, 4, 5] these two common conditions continue to be confused with each other. They represent two separate and distinct entities. Verruca senilis is a benign epithelial nevus, whereas keratoma senile belongs in the group of precancerous dermatoses.

Verruca Senilis (Seborrheic Keratosis)

Verruca senilis usually develops in the later decades of life as multiple lesions. These verrucæ vary greatly in size and occur chiefly on the trunk in the so-called seborrheic areas, although at times they appear in large numbers on the face also (Fig. 1*a*). They are not seen on the mucous membranes, the palms, or the soles. The lesions usually begin as small, light-yellow areas which increase in size and become more deeply pigmented, so that many of them are deep brown or even bluish brown when fully developed. They are usually covered with a thin greasy scale which is readily removed by gentle friction; they have the appearance of being "stuck on" the skin. A granular-appearing surface frequently results due to small plugs. When the lesions are elevated or pedunculated and deeply pigmented, they may be confused with benign pigmented nevi (Fig. 1*b*). The latter, however, do not have a soft, greasy plug. When the lesions occur on the face, many may show relatively little pigment and have a warty character, but even here the crust is softer and the lesions lack the induration palpable in keratoma

*From the Section on Dermatology and Syphilology, The Mayo Clinic, Rochester, Minnesota.

senile; nor is there any inflammatory reaction around the lesion, as is commonly the case in keratoma senile. Occasionally it may be necessary to take a specimen for biopsy in order to differentiate the early, non-elevated verrucous form of verruca senilis from an early lesion of keratoma senile. Because of the deep brown, almost black, appearance of some of the lesions, at times they have been excised on the basis of being melanomas. The latter have a slate blue or slate gray color and lack the other features of verruca senilis. Nevus blau (blue nevus of Jadassohn) presents a smooth surface without scales and a bluish rather than brownish pigmentation. Dermatosis papulosa nigra,[4] occurring among negroes, although developing earlier in life, is merely a variant, I believe, of verruca senilis and presents a similar histologic picture.

Fig. 1. Verruca senilis: *a*, This woman, aged sixty-one years, had had the lesions for ten years; there were also many lesions on the trunk. Biopsy of two borderline lesions confirmed the clinical impression that all lesions in this case were verruca senilis, and that none were keratoma senile. The variation in size and elevation of the lesions will be noted. *b*, This deeply-pigmented elevated lesion back of the ear should be differentiated from a benign pigmented nevus. *c*, Keratoma senile: The discrete lesions and plaques on the face, forehead, and ears may be noted, as well as the epithelioma, which arose, presumably, from a senile keratoma; histologically this lesion proved to be of the squamous cell variety, grade 2, with individual cell keratinization.

Verruca senilis, histologically (Fig. 2*a*), shows a varying picture whether the tumor is elevated or verrucous in character; in all types, however, there is definite cyst formation which is not traceable as a rule to the hair follicles or sebaceous glands but represents invagination of the epidermis. There is marked pigmentation of basal cells, and also acanthosis and prolongation of the rete ridges and a definite regular cycle of keratinization which result in epithelial cysts. There are relatively few mitiotic figures, and usually there is little inflammatory reaction in the cutis. The lesions should not be confused microscopically with pigmented basal cell epithelioma or with tricho-epithelioma (epithelioma adenoides cysticum).[5]

Whereas there have been occasional reports of malignancy occurring in verruca senilis,[6] usually basal cell in type, in my experience this has happened only when the lesion has been subjected to repeated irritation or trauma. Verruca senilis must be regarded as a benign dermatosis, probably a delayed epithelial nevus, and need be removed only for cosmetic purposes or when located in areas subject to trauma or irritation. As the lesions do not extend below the level of the

epidermis, they may be removed by fulguration or cautery as well as by excision. They do not respond well to radiotherapy because of the marked degree of differentiation of the individual cells.

epithelioma in situ and showing all the microscopic features of Bowen's disease. As a rule, very little pigment is seen in the basal cells, and there is definite infiltration of cells in the cutis. Malignant change in one or more of the lesions

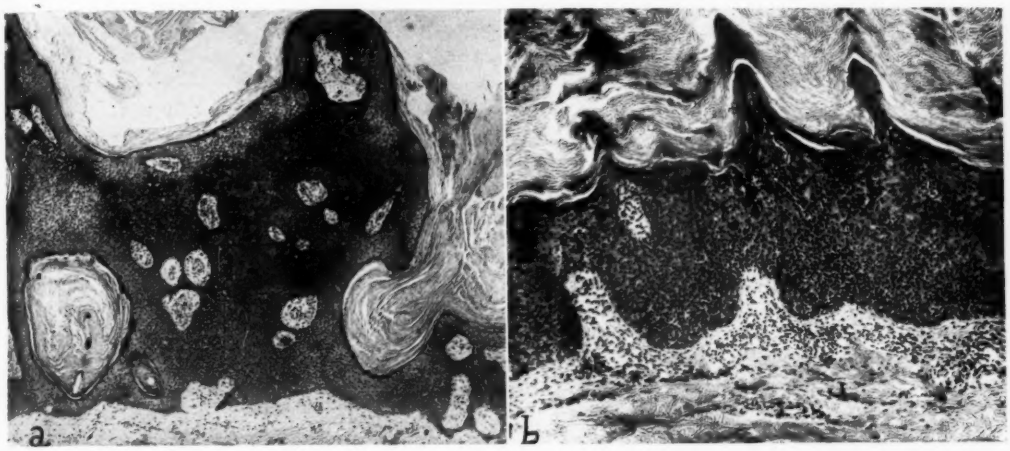

Fig. 2. *a*, Verruca senilis: Section of lesion in Figure 1 *b*, showing mixture of verrucous and solid forms. The deposits of melanin pigment and cyst formation may be noted. (Silver nitrate and hematoxylin stains.) b, Keratoma senile: The infiltrate in the cutis, lack of pigmentation, and early phenomena of dyskeratosis of the cells of the epidermis may be noted. (Hematoxylin and eosin stains.)

Keratoma Senile

Keratoma senile (Fig. 1*c*) also occurs in elderly individuals and the lesions are found chiefly on the exposed surfaces of the body, especially among persons who have a sandy complexion and who are frequently exposed to the elements (sailors' skin). The lesions may, however, be found on any part of the body. They appear as discrete, sharply circumscribed, flat, elevated or verrucous, brown to grayish, keratotic papules, or as ill-defined, scaling patches. The scales tend to be adherent, and their removal may result in points of bleeding. There is an appreciable induration to the lesions on palpation and often a zone of erythema surrounds a papule or plaque. When the latter is present, it is frequently indicative of early malignant change. Occasionally, keratoma senile develops into a cutaneous horn (cornu cutaneum).

The histologic picture of keratoma senile is much less distinctive than that of verruca senilis. In the early stages of the disease it may resemble that of an ordinary wart, but, shortly, various phenomena of dyskeratosis (Fig. 2*b*) and individual cell keratinization are seen in the prickle cell layer, eventuating in many instances in an epithelioma in situ and showing all the microscopic features of Bowen's disease. As a rule, very little pigment is seen in the basal cells, and there is definite infiltration of cells in the cutis. Malignant change in one or more of the lesions occurs in from 20 to 25 per cent of the cases of keratoma senile, and, when epithelioma is present, in more than 90 per cent of the cases it is squamous cell in character and varies from grade 1 to grade 4 in malignancy. There may be microscopic evidence of epithelioma in situ without clinical evidence of the lesion being epitheliomatous, and the lesion may persist for many years without developing into an invading squamous cell epithelioma, hence the inclusion of keratoma senile among the so-called precancerous dermatoses. In a few instances keratoma senile histologically reveals prolongation of the rete ridges in narrow strands, and it is in these instances that one may anticipate the development of basal cell, rather than squamous cell, epithelioma.

Keratoma senile should be destroyed by fulguration, surgery, or excision, with the realization that the lesion may extend deeply heneath the epidermis and is not "stuck on" as is the lesion of verruca senilis. Because lesions of keratoma senile histologically frequently simulate the picture of Bowen's disease, which picture is also frequently present in early epithelioma from roentgen rays, treatment with roentgen rays or radium is contraindicated.

Bibliography

1. Eller, J. J., and Ryan, V. J.: Senile keratoses and seborrheic keratoses; differences clinically, histologically and in susceptibility to malignant change. Arch. Dermat. and Syph., 22:1043-1060, (Dec.) 1930.
2. Freudenthal, Walter: Verruca senilis und Keratoma senile. Arch. f. Dermat. u. Syph., 152:505-528, 1926.
   Freudenthal, Walter, and Spitzer, R.: II. Warzen und Kondylome: Geschwülste der Haut. In: Jadassohn: Handbuch der Haut und Geschlechtskrankheiten. Berlin, Julius Springer, pp. 33-207, 1933.
3. Hookey, J. A.: Keratoma senilis and verruca senilis; clinical and histopathologic study. Arch. Dermat. and Syph., 23:946-959, (May) 1931; Proc. Staff Meet., Mayo Clinic, 5:239-241, (Aug. 27) 1930.
4. Michael, J. C., and Seale, E. R.: Dermatosis papulosa nigra. Arch. Dermat. and Syph., 20:629-640, (Nov.) 1929.
5. Montgomery, Hamilton, and Dörffel, J.: Verruca senilis und Keratoma senile. Arch. f. Dermat. u. Syph., 166:286-296, (Sept.) 1932.
6. Pinkus, F.: Über einen Fall von Basalzellenepitheliom auf Verruca senilis. Arch. f. Dermat. u. Syph., 169:58, (Sept.) 1933.

ESSENTIAL HYPERTENSION AND TUBERCULOSIS

CARL G. MORLOCK, M.D.†

Rochester, Minnesota

THE study of arterial hypertension in pulmonary tuberculosis has attracted attention for a long time. Marfan in 1891 reported that hypotension was the most common finding in tuberculosis, and reaffirmed this in 1907.[10] He said that a normal or habitually elevated blood pressure had a favorable prognosis in chronic pulmonary tuberculosis, whereas a continuing low blood pressure was of poor prognosis. He also cited the finding of hypotension as an aid in the diagnosis of pulmonary tuberculosis, and considered it a valuable observation in conjunction with an unexplained albuminuria in making a diagnosis of renal tuberculosis in the presence of a known tuberculous focus. Pottenger[14] and Bethea[2] reached similar conclusions. Fishberg[8] said that phthisic subjects have a blood pressure that is lower than normal. In a series of 464 cases of pulmonary tuberculosis, Stivelman[18] found a hypotension in 90 per cent, and a normal blood pressure in the remaining 10 per cent. In this latter group the mean systolic pressure did not exceed 135 mm. of mercury. Glaros[9] found that regardless of the clinical course of the disease, the blood pressure in pulmonary tuberculosis was normal or below normal in 99 per cent of cases. Brelet[3] cited a case in which the blood pressure was extremely variable. The systolic blood pressure ranging between 240 and 70 mm. of mercury and the diastolic blood pressure varying between 140 and 45 mm. of mercury. The ultimate diagnosis in this case was not made until necropsy revealed an extensive caseous pulmonary tuberculosis. Brelet explained the hypotensive exacerbations by assuming a depressant action on the vasomotor mechanism by the toxins of the tubercle bacilli.

In renal tuberculosis, opinions of writers regarding blood pressure observations correspond to those with reference to pulmonary lesions. Pousson and Desnos[15] said that in renal tuberculosis the blood pressure on the whole is normal or below normal, though a minor hypertension may coexist. Reitter[16] reported ten cases of known renal tuberculosis and in six of the cases the systolic pressure was 90 mm. of mercury or lower, in two cases it was normal, and in one case there was a definite hypertension, the systolic pressure being 160 mm. of mercury. Reitter[16] believed that in cases of gross urinary infection, a coexistent hypotension could be used as an aid in establishing the tuberculous nature of the lesion. Elaut[7] on the other hand, did not believe that the blood pressure curve was dependent on the presence of renal tuberculosis. In a review of twenty cases, he found that the average values for arterial tension were 145 mm. of mercury for the systolic pressure and 70 mm. of mercury for the diastolic pressure.

Barbier and Cibert[1] reported a case of active

†Fellow in Medicine, The Mayo Foundation, Rochester, Minnesota.

unilateral renal tuberculosis in which the systolic blood pressure was 240 mm. of mercury and the diastolic pressure was 100 mm. of mercury. In following this case after nephrectomy, within one month they found a reduction of arterial tension to 170 mm. of mercury for the systolic, and 100 mm. of mercury for the diastolic blood pressure.

Morlock and Horton[12] have studied a much larger series of cases of renal tuberculosis at The Mayo Clinic. In a critical review of 346 cases of proved renal tuberculosis, in which nephrectomy was carried out, it was found that the vast majority of subjects (approximately 78 per cent) had normal blood pressures; 20 per cent had hypertension, that is, a systolic reading of 140 mm. of mercury or above; and approximately 2 per cent had hypotension, that is, a systolic blood pressure below 100 mm. of mercury. For the various age groups, we have regarded a systolic pressure which ranges from 100 to 140 mm. of mercury as being normal. The ages of the subjects ranged from eleven to sixty-eight years. The number of subjects in this series who had hypotension and hypertension is probably not any greater than one would expect to find in a control series of so-called normal subjects in the same age groups. While making this study, the following case was observed and seems worth reporting in detail because of the association of relatively severe hypertension with clinically healed renal and pulmonary tuberculosis.

A man, forty-seven years of age, presented himself at the clinic on October 2, 1934, for advice regarding hypertension, which he had been cognizant of for ten years. When he first consulted a physician elsewhere ten years ago, a systolic blood pressure of 200 mm. of mercury was noted. His family history is of interest. His grandmother (side of family tree not stated in history) had had hypertension, his father had died of "acute indigestion," his mother, who is still living, had tuberculosis (apparently pulmonary) and now has hypertension. Twenty-six years ago he had first sought medical advice elsewhere because of dyspnea, urinary frequency and urgency, and hematuria. At that time, examination had revealed evidence of bilateral active pulmonary tuberculosis, with fever and general malaise. Cystoscopic examination had disclosed an ulcerated area at one ureteral orifice (he did not recall which one) and a diagnosis of tuberculosis of the bladder had been made. A regimen of rest had been prescribed, a high caloric diet had been recommended and the bladder had been treated locally. In six months he had improved to such an extent that he had returned to work, although the vesical symptoms had continued for ten years. During the past fifteen years he had had no urinary symptoms except transient burning on

Fig. 1. *a*, Extensive calcification of the right kidney; *b*, right ureter; *c*, discrete areas of calcification in the region of the prostate.

micturition, which had required no treatment. His history otherwise did not reveal anything abnormal.

On examination he was found to be a well-nourished man, apparently in good health, whose height was 69 inches, and whose weight was 184 pounds. His temperature was 98.6° F., the pulse rate was 76 per minute, and the systolic blood pressure was 205, and the diastolic was 130 mm. of mercury. The pupils reacted to light and accommodation. The teeth were in good condition except for a mild degree of pyorrhea. The tonsils had been removed elsewhere. Examination of the chest revealed moderate emphysema and scattered moist râles in both apices on forced inspiration. The heart rate and rhythm appeared normal. The apical impulse was situated in the left fifth interspace, 10 cm. from the midsternal line, the aortic second sound was accentuated, grade 1+. The abdomen was normal; examination of the genitalia revealed bilateral testicular atrophy. The deep tendon reflexes were normal. Palpation of the peripheral vessels disclosed a rubbery type of sclerosis, grade 2. Examination of the ocular fundi showed generalized arterial narrowing 1+ of the hypertensive sclerotic type; the disks were normal.

The concentration of hemoglobin was 18.5 gm. per

100 c.c. of whole blood (by photo-electrometer), and the leukocytes numbered 5,200 per cubic millimeter of blood. The Wassermann reaction of the blood was negative. The value for urea was 26 mg. per 100 c.c. of

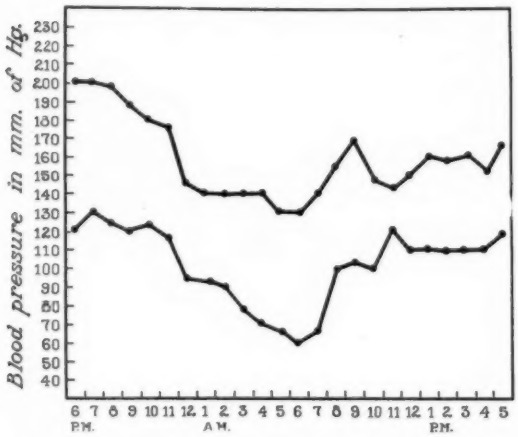

Fig. 2. Record of hourly blood pressure taken over a period of twenty-four hours while the patient was at rest. Fluctuations of this type are usually observed in cases of essential hypertension.

whole blood. The urine was normal; the specific gravity was 1.026, and the reaction was acid. Roentgenograms of the thorax revealed evidence of healed pulmonary tuberculosis of both upper pulmonary fields. Roentgenograms of the kidneys, ureters, and bladder (Fig. 1) disclosed a large calcified mass in the right renal area, an elongated area of calcification opposite the last lumbar vertebra on the right side (calcified ureter), and small discrete areas of calcification in the region of the prostate. The electrocardiogram was normal except for slight left ventricular preponderance.

When the blood pressure was taken at hourly intervals over a period of twenty-four hours, while the patient was at rest, the systolic pressure ranged from 130 to 200 mm. of mercury and the diastolic pressure varied from 60 to 130 mm. of mercury (Fig. 2). Fluctuations of blood pressure of this degree are frequently observed in subjects with essential hypertension.

In a review of the pertinent literature on renal tuberculosis, one finds a general note of warning as to the seriousness of this condition. Wildboltz[19] noted that in 316 cases there was a mortality of 58 per cent within five years, and that only 6 per cent of these patients lived more than ten years. Persson[13] reported a mortality of 84.5 per cent within ten years of all cases in which nephrectomy was not carried out. He cited the records of two patients who were both clinically well after periods of seventeen and twenty-seven years, respectively. Caulk[4] and Persson[13] were of the opinion that it is impossible to demonstrate spontaneous healing of renal tuberculosis, though they admit clinical arrest by a process they term autonephrectomy. Medlar[11] proved to his own satisfaction that, in experimental animals, artificially produced renal tuberculosis could heal. D'Abreu[6] felt that eight years' quiescence of renal tuberculosis was notable. Christian and Vintinci[5] and Schall[17] each reported a single case in which complete calcification of the kidney, as revealed by the roentgenogram, apparently had resulted in arrest of the tuberculous process.

In consideration of the case presented, there are two notable features. First is the presence of a relatively severe essential hypertension in a case in which the patient had rather extensive clinically arrested tuberculous lesions in the lungs, right kidney, ureter, and prostate. Second is the apparent complete calcification and clinical arrest of a renal tuberculous lesion twenty-six years after a primary diagnosis.

Bibliography

1. Barbier, J., and Cibert, J.: Tuberculose rénale ouverte avec hypertension. Néphrectomie. Guérison clinique rapide et heureux effet de l'intervention sur hypertension. Lyon méd., 147:441-446, (Apr. 5) 1931.
2. Bethea, O. W.: Blood-pressure in pulmonary tuberculosis. New Orleans Med. and Surg. Jour., 81: 314-316, (Nov.) 1928.
3. Brelet, M., and Perrin, P.: Hypertension artérielle et tuberculose. Gaz. de Hôp., 101:469-479, (Mar. 28) 1928.
4. Caulk, J. R.: Renal tuberculosis. Jour. Urol., 6:97-113, (Aug.) 1921.
5. Christian, E., and Vintinci, V.: Un cas de rein tuberculeux calcifié exclu. Jour. d'urol., 34:219-221, (Sept.) 1932.
6. D'Abreu, A. L.: Closed renal tuberculosis, with special reference to the possibility of healing this disease. Brit. Med. Jour., 1:605-606, (Apr. 8) 1933.
7. Elaut, L.: La tension artérielle chez les urinaires. Jour. d'urol., 29:441-458, (May) 1930.
8. Fishberg, Maurice: Pulmonary tuberculosis. Philadelphia: Lea and Febiger, Ed. 3, pp. 281, 1922.
9. Glaros, Constantin: Tension artérielle et tuberculose. Presse méd., 2:1579-1581, (Dec. 12) 1928.
10. Marfan, A. B.: Tuberculose pulmonaire chronique et son importance pour le pronostic. Rev. de méd., 27:1005-1022, 1907.
11. Medlar, E. M., and Sasans, K. T.: Experimental renal tuberculosis: with special reference to excretory bacilluria. Am. Rev. Tuberc., 10:370-383, (Dec.) 1924.

12. Morlock, C. G., and Horton, B. T.: Variations in blood pressure in renal tuberculosis. Proc. Staff Meetings of Mayo Clinic, 10:101-102, (Feb. 13) 1935.
13. Persson, Mauritz: Renal tuberculosis: a clinical survey of 295 cases, 90 of which were not operated on. Ann. Surg., 82:526-551, (Oct.) 1925.
14. Pottenger, F. M.: The effect of tuberculosis on the heart. Arch. Int. Med., 4:306-322, (Oct.) 1909.
15. Pousson, A., and Desnos, E.: Encyclopédia Française d'Urologie. Paris: Octave Doin et Fils. 2: pp. 764, 1914.
16. Reitter, Karl: Nierentuberculose und arterielle Hypotension: Ein differentialdiagnostisches Symptom von praktischer Bedeutung. Ztschr. f. klin. Med., 62:359-367, 1907.
17. Schall, L.: Die Mörtelniere, Endstadium einer kindlichen Nierentuberkulose. Deutsch. med. Wchnschr., 2:1975-1977, (Nov.) 1931.
18. Stivelman, B. P.: Observations on the blood pressure in pulmonary tuberculosis. Am. Jour. Med. Sc., 173:46-59, (Jan.) 1927.
19. Wildboltz: Quoted by Eisendrath, D. N., and Rolnick, H. C.: In: Text-book on urology. Philadelphia: J. B. Lippincott and Company, pp. 717, 1928.

HERPES ZOSTER OPHTHALMICUS*

PAUL D. BERRISFORD, M.D.
Saint Paul

WHETHER herpes zoster is a clinical entity has occupied the minds of clinicians for some years. The etiology of a group of infectious diseases characterized by vesicular eruptions such as sheep pox, horse pox, cow pox, varioloid, alastrim, chicken pox, herpes zoster, herpes simplex and epidemic encephalitis, is, in this regard, called into question. While one in the group may resemble another, others show nothing in common. Experimental evidence is not as yet forthcoming to prove their kinship. In this regard, experimental research has led to the conclusion that herpes zoster ophthalmicus has no common relationship with dendritic or stellate keratitis.

There has appeared in the literature much dealing with the association of herpes zoster with varicella. Le Feuvre[6] holds that herpes zoster may, when it occurs under certain conditions, be the start of a chicken pox epidemic. Savin[10] contends that either disease may cause the other, the causal factor having two phases, one generalized causing varicella, the other localized and giving rise to herpes zoster.

Although herpes zoster is included among those diseases caused by a filtrable virus, there is no direct evidence that such is the case. That it is due, in all probability, to a filtrable virus is inferred by the fact that every attempt to find a culturable bacterium has ended in failure. In addition, there are found nuclear inclusions in the epithelial cells of the lesions similar to those produced by the virus of herpes febralis.

However some progress has been made experimentally. Netter and Urbain,[9] Bedson and Bland,[2] Aitken and Brain,[1] have shown that the serum of herpes zoster convalescents contains specific antibodies whose presence can be detected by the complement-fixation reaction, using, as antigen, diluted zoster vesicular fluid or a suspension of crusts of the zoster skin lesion. The most that can be said at the present time is that herpes zoster is the result of a specific substance, which, when the disease has run its course, leaves evidence of its former existence through the presence of antibodies in the serum.

Attempts to transmit the disease to animals have proven a failure. Cole and Kuttner[3] of the Rockefeller Institute, working with rabbits, guinea pigs and monkeys, met with no success. They have shown, however, that the disease is contagious, a fact suggested prior to their work by repeated epidemic outbreaks, especially during the summer and late fall. As proven by the above named workers, one attack of herpes zoster usually confers immunity.

As far back as 1862 the pathologic condition in herpes zoster, while not definitely known, was correctly surmised. Campbell and Head,[4] in 1900, exhaustively studied seventeen cases the duration of which varied from a few days to one and one-half years and in all cases there were found hemorrhages in the posterior root

*Read at the meeting of the Miller Hospital Staff, February 5, 1935.

ganglia, usually small and surrounded by considerable inflammatory exudation. The ganglion cells were destroyed in varying extent and in the late cases the ganglia were markedly sclerosed. Where herpes zoster affected the area supplied by the opththalmic division of the fifth nerve, all showed the lesion to be in the Gasserian ganglion.

General paralysis, tabes, the prolonged use of arsenicals, influenza, the administration of antityphoid vaccine are said to be predisposing factors in the production of the disease. Herpes zoster occurs mostly in the middle-aged, occasionally in children. In the aged it takes on a more virulent form.

As a rule there is a prodromal period of three or four days antedating the rash. Over the area of future vesication there generally occurs burning or lancinating pains. During the period of hyperesthesia, temperature is usually present; then follow the stages of erythema, vesication and rupture of the vesicles, cicatrization and finally anesthesia. In some cases the pain is very severe and the absence of some degree of anesthesia is infrequent.

The ophthalmic division of the fifth nerve before entering the superior orbital fissure gives off a branch to the meninges, the involvement of which may cause intense headaches and vomiting. The naso-ciliary nerve innervates large areas of the mucous membrane of the nasal septum and the outer wall of the antrum; its involvement in the process gives rise to most disagreeable nasal symptoms. It is affected in about 50 per cent of cases.

Foster Moore[8] states that ocular complications such as corneal involvement, iridocyclitis, ocular palsy or optic atrophy, follow in the wake of the eruption in herpes zoster ophthalmicus in 60 per cent of cases.

There is usually hyperemia of the conjunctiva, not seldom pronounced conjunctivitis. When the cornea is involved the disease takes on protean forms. It may be the seat of patchy interstitial inflammation, or the appearance of the cornea may simulate a dendritic keratitis, gray striæ extending in one or more directions from the minute ulcer, throwing off branches and finally breaking down. Large ulcers may form, enlarge and fuse, with considerable denudation of the epithelium, leaving a craterlike border. These ulcers are always superficial unless they become infected, when they then extend into the depths, producing an hypopion. They are characteristically chronic and are prone to relapse. Occasionally the surface of the cornea is raised to form vesicles and, later, small ulcers.

Iridocyclitis frequently appears alone or associated with corneal involvement. Secondary glaucoma may result although hypotension is the more frequent accompaniment of this disease.

Hunt[5] collected 158 cases of paralyses of various parts of the body which occurred in herpes zoster and, of these, eighteen showed paralysis of the third nerve. Metz[7] collected fifty case of ocular palsy accompanying herpes zoster and found the third nerve partially or completely paralyzed in 74 per cent of them. It has been observed in most cases of this nature that there is a partial or eventually a complete recovery.

In the few cases of optic atophy reported as the result of herpes zoster, no previous ophthalmoscopic examination had been made, thus rendering herpes zoster, as the etiological factor, questionable.

The explanation of the ocular disease on the basis of the site of the lesion meets with difficulties. We regard sensory nerves as conveying afferent impulses only. Why a block in the course of the nerve should produce trophic changes in structures at its distal terminations is not clear. While it is true that scrupulous care exercised in preventing mechanical injury to the desensitized cornea, even suturing the eyelids together, may prevent in many cases corneal involvement, nevertheless such measures may prove of no avail. However, we know that section of motor nerves produces trophic changes in skeletal muscles, which becomes atrophic and finally are reduced to connective tissue. Nevertheless, as stated by Starling,[11] "Whether we are justified in assuming the existence of trophic nerves exerting an influence on the part they supply apart from any influence on its other functions, experimental evidence is not sufficient to decide." It would appear, then, that any interference of sufficient degree with motor or sensory physiological activity of a part may produce trophic changes. To assume that sensory nerves are capable of antidromic impulses is untenable.

The treatment of the disease should be symp-

tomatic and along the lines generally employed for iridocyclitis and corneal affections. For pain, the salicylates or morphine or its derivatives, hot fomentations locally, holocaine, et cetera, are indicated.

In reviewing articles dealing with the treatment of herpes zoster the writer finds the following therapeutic agents recommended: autoserotherapy, the high frequency current, deep x-ray radiation, ultra-violet ray, sodium iodide, arsphenamine, neoarsphenamine, sulpharsphenamine, et cetera. The author believes that herpes zoster is a self-limited disease for which the above named therapeutic agents have no special specificity, indeed little if any value. The condition when first seen is usually in full bloom, with a fully established focal pathology, and nature has already started the patient on the road to recovery.

The most that we can hope to do is to keep the patient as comfortable as possible through the relief of pain, and so guide the course of the eye affection as to avoid, if possible permanent ocular impairment.

Case Reports

Case 1.—Mr. E. E. C. of Deer River, Minn., a material checker, while weighing a load of cement, thought a gust of wind had carried some of the material into his eyes. Four days later he consulted the local physician because of pain in the corner of his right eye. Ten days later blisters appeared over the right side of the forehead and the right eyelids.

When seen, sixteen days after the alleged accident, the patient complained of reduced vision in the right eye as well as a constant burning, smarting and itching of the affected eye. Ophthalmologic examination showed the vision of the right eye to be 20/40ths. Bulbar and tarsal conjunctival infection was present, with passive congestion of eyelids. There were deep ciliary injection and fibrin deposits on the posterior surface of cornea. The pupillary border of the iris was almost completely bound down to the anterior capsule of the lens, and there were fibrin deposits on the anterior surface of the lens in the pupillary area. The fundus could not be seen owing to the fibrin deposits on the posterior surface of the cornea, and the anterior surface of the lens, and vitreous opacities. Ocular tension on palpation appeared normal. The cornea was absolutely anesthetic. Over the right side of the forehead were blotchy areas of skin pitting, evidence of former vesication. The skin showed marked reduced sensibility over the forehead and upper right eyelid.

The patient was given three intravenous injections of triple typhoid vaccine during the course of a week, sun-lamp treatments daily, and, locally, hot fomentations and the usual regime for iridocyclitis. He was discharged after ten days in the hospital.

A month later there was a marked dermatitis involving the right eyelids and contiguous parts, the skin being dusky red, encrusted and very irritated. The iridocyclitis had greatly subsided but the pupillary border was free only "in notches." Vision was corrected to 20/40ths.

Fig. 1. Case 2 (see text).

After a few days in the hospital, the patient developed pain in the right fingers followed soon after by the appearance of large serous blebs over the affected parts. After remaining again ten days in the hospital he was discharged, the iridocyclitis having completely subsided but the corneal anesthesia still remaining. Vision with correction 20/40ths.

Case 2.—Mr. F. A. H., pattern maker, aged twenty-six, stated that four days previous he was taken with a pain in the right cheek, right forehead and over the right side of the nose. Serous blebs were linearly distributed in a perpendicular direction over the skin of the right forehead, over the surface and the tarsal border of the upper right eyelid and on the right side of the nose. The bulbar and tarsal conjunctivæ were injected and the cornea showed relative anesthesia.

Nine days later ciliary injection appeared and with it the presence of a small linear ulcer near the corneal center. The corneal lesion showed gradual improvement only to be followed by an exacerbation six days later when there appeared several cup-shaped, tiny ulcers distributed over the corneal surface. Three weeks later the corneal lesions had completely healed

but there still remained relative anethesia of the cornea as well as the skin of the right forehead, and right upper eyelid. The vision was unimpaired but the forehead was left deeply pitted.

Case 3.—Mr. O. T., aged thirty-four, a brewery employee, was exposed to wide ranges of temperature, being constantly in and out of a refrigerator. Three days previous "he thought he got something into his left eye." About this time an eruption broke out on the left side of his forehead attended with swelling beneath his left eye. He gave no history of neuralgic pains.

Examination disclosed a linear group of vesicles coursing perpendicularly up the skin of the left forehead with two blebs involving the left upper eyelid. In addition there was an edematous patch beneath the left eye.

There existed a moderate degree of conjunctival congestion, a relative anesthesia of the left cornea but no other ocular involvement. Convalescence was uneventful.

Case 4.—Mr. O. M., high school teacher, aged fifty-five. Two weeks previous the left eye felt as though a foreign body were in it and this feeling had persisted. About five days later the left side of forehead showed diffuse swelling with a patch of edema involving the inner third of the left eyebrow and a large patch immediately below the left eye. Two days later the forehead showed over its congested surface a studding of vesicles.

The left forehead, left eyelids and left cornea showed lessened sensibility to touch. In the center of the cornea were tiny areas of superficial infiltration with a moderate degree of circumcorneal injection. The ocular pain, lachrymation and photophobia were intense.

After the subsidence of the ocular symptoms neuralgic pains over the left forehead persisted for some weeks and a relative anesthesia of the left cornea and forehead.

Bibliography

1. Aitken, R. S., and Brain, R. T.: Facial palsy and infection with zoster virus. Lancet, 1:19-22, (Jan. 7) 1933.
2. Bedson, S. P., and Bland, J. O. W.: On supposed relationship between viruses of herpes febrilis and vaccinia. Brit. Jour. Exper. Path., 9:174-178, (Aug.) 1928.
3. Cole, R., and Kuttner, A. G.: Etiology of herpes zoster. Jour. Exper. Med., 42:799-820, (Dec.) 1925.
4. Head, H., and Campbell, A. W.: The pathology of herpes zoster and its bearing on sensory localization. Brain, 23:353-523, 1900.
5. Hunt, J. R.: On herpetic inflammations of the geniculate ganglion. A new syndrome and its complications. Jour. Nerv. and Ment. Dis., 34:73-96, (Feb.) 1907.
6. Le Feuvre, W. P.: A plea for the recognition of a common origin for shingles and chickenpox. Brit. Jour. Dermat., 29:256-262, (Oct.) 1917.
7. Metz, R. B.: Herpes zoster ophthalmicus complicated by ophthalmoplegia. Ohio Med. Jour., 9:590-593, (Dec.) 1913.
8. Moore, R. F.: Ocular manifestations of lesions of fifth cranial nerve. Brit. Med. Jour., 2:783-786, (Oct. 29) 1932.
9. Netter, A., and Urbain, A.: Nouvelles recherches sur la déviation du complément dans le zona. L'antigène du zona n'exerce aucune action sur le sérum des sujets atteints d'herpes. Comp. rend. Soc. de biol., 90:461-464, 1924.
10. Savin, L. H.: Discussion of relationship, direct or indirect, between certain cases of herpes ophthalmicus and varicella. Tr. Oph. Soc. U. Kingdom, 53:271-281, 1933.
11. Starling, E. H.: Principles of Human Physiology. Philadelphia, Lea and Febiger Co., 1920, 3rd ed., p. 349.

SOME OBSERVATIONS ON THE INTRANASAL OPERATION FOR FRONTAL SINUSITIS*

CARL M. ANDERSON, M.D.

Rochester, Minnesota

IN a recent investigation to determine the principal causes of failure of operations for the relief of frontal sinusitis, it was found that a large percentage of failures were the result of incomplete intranasal operations and a collapse of the soft parts into the frontal sinus and nasofrontal duct, in cases in which the bony wall of the sinus had been removed by an external operation.[1] In nearly all cases that were reviewed, the immediate result of operation seemed good; all symptoms subsided promptly, and one was led to believe that the cure would remain permanent. After several months, and in some instances after several years, there was a return of symptoms; this probably was the result of reinfection; but some form of obstruction to

*From the Section on Otolaryngology and Rhinology, The Mayo Clinic, Rochester, Minnesota. Read before the Minnesota Academy of Ophthalmology and Otolaryngology, at Rochester, Minnesota, April 12, 1935.

proper drainage had taken place, which prevented the prompt resolution of the acute process.

The conclusion was reached at that time that, if a method could be devised by which the bony structure, which surrounds the frontal sinus and the nasofrontal duct, could be preserved, and adequate drainage provided, the result probably would be more ideal than it had been. The failures with the intranasal operations appeared to be the result of incomplete removal of all the ethmoid cells, in the neighborhood of the nasofrontal duct, and the fronto-ethmoid cells, which may extend well up over the orbital wall. These cells often take up space which otherwise would be included in the frontal sinus. In many instances, the roof of a large fronto-ethmoid cell forms the floor of the frontal sinus.

In an effort to overcome the difficulties, which occur in many cases, as a late result of the external operation, and to provide an adequate nasofrontal duct that would remain patent, a great deal of attention was paid to the complete removal of all the cells in this region, by the intranasal route. In order to facilitate the removal of the anterior cells, it has been found necessary to flatten the agger nasi and to remove completely any cells which may be in it. A fairly large portion of the anterior end of the middle turbinate bone may have to be removed in order to provide for the complete removal of the bulla ethmoidalis and the uncinate process. If any of these structures are left in place, the approach is much more difficult, and there is a deep excavation with somewhat perpendicular walls which will probably fill in with granulations, instead of a rather large concave area with gently sloping sides, without anything to interfere with direct drainage of the frontal sinus into the nose.

In approaching any surgical condition one must always be prepared to meet each problem presented with the type of operation best suited to the individual or pathologic condition encountered. In the treatment of disease of the frontal sinus, as in the treatment of diseases elsewhere in the body, no one procedure can be considered to displace any other method entirely. One will always encounter cases in which necrosis of the bony structures has taken place, or polyps and degenerated membranes exist inside of the sinus to such an extent that an approach through the external wall becomes necessary in order to eradicate the disease completely. In a number of cases in which it seemed necessary to perform an external operation, drainage by the intranasal route proved entirely adequate to control the disease.

Fig. 1. Large dilator passed directly into the frontal sinus.

It would seem that the approach through the orbital wall, as advocated by Sewall and others, would, to a large extent at least, overcome the tendency of the soft parts to obstruct the frontal sinus and nasofrontal duct. With this type of operation, the periosteum lining the orbital cavity is dissected off and retracted with the orbital contents in order to expose the partition between the orbit and labyrinth of the ethmoid. This should restrain the tendency of the soft parts to bulge into and fill the cavities produced by the removal of bone.

In cases in which operation has been performed previously or in which infection has been present for a long time and has produced a regeneration of bone or thickening of the bones as a result of osteitis, the external approach is the method of choice.

Before about 1890, the approach to the frontal sinus had been by the external route. At this time, Schaeffer advocated the draining of the frontal sinus by enlarging the nasofrontal duct. About 1891, Winkler and Scheier demonstrated the relation of the frontal sinus to other structures in the upper portion of the nose and proved that it usually was practical to approach the frontal sinus by the intranasal route. In 1893, Jansen called attention to the great importance

of cleaning out the anterior ethmoid cells thoroughly, and of removing the anterior portion of the middle turbinate bone in order to secure the largest duct and to maintain adequate drainage.

Fig. 2. *a*, Bony structure around the nasofrontal duct and the fronto-ethmoid cell, forming the floor of the frontal sinus; *b*, probe passed into the nasofrontal duct lateral to the fronto-ethmoid cell, and *c*, fronto-ethmoid cell broken down, providing direct drainage into the nasal cavity.

In a review of the anatomy of the middle turbinate bone, anterior ethmoid, and fronto-ethmoid cells, one finds that the nasofrontal duct is under the anterior end of the middle turbinate bone (Fig. 1) between the uncinate process and the bulla ethmoidalis. In this region one will often encounter several layers of cells. A rather deep cavity is formed laterally after the removal of these cells. In a large number of instances one encounters a large fronto-ethmoid cell, which extends up over the orbital roof. The superior wall of the cell forms the floor of the frontal sinus. In probing the frontal sinus (Fig. 2, *a*, *b* and *c*) this cell may readily be mistaken for the sinus. For the intranasal approach to the frontal sinus, anteroposterior and lateral roentgenograms are very essential. The lateral view will show the anteroposterior depth of the frontal sinus and the anteroposterior view will reveal the space available between the septum and the lateral wall of the nose. It also will show the relative position of the frontal sinus to the neighboring cells, particularly the fronto-ethmoid cells.

If there is any deflection of the nasal septum, it should be taken care of first, because if it is left in place it would interfere with the surgical approach and later might prevent proper postoperative treatment. The anterior third of the middle turbinate bone should then be removed by means of scissors or biting forceps. Following this, the agger nasi should be flattened with biting forceps; the flattening process should be completed with backward biting forceps. A modified Kerrison punch has been found to be very convenient for this purpose. It is very strong, takes a shallow bite, and allows one to work from above, and to exert what pressure is needed, downward and forward. In this way, a much better view can be obtained as well as an easier approach to the anterior ethmoid cells which surround the nasofrontal duct. Often, rather large cells are found in the agger nasi, which, if left, would spoil an operation which otherwise would be well done. Occasionally, one encounters rather dense bone in this region and may be forced to break through the mesial wall with a curet. Attention should be called to the proximity of the lacrimal bone and the lacrimal gland. These structures are situated slightly lower than the agger nasi, and in my experience they have not been injured in this type of operation. The bulla ethmoidalis, uncinate process, and anterior ethmoid cells should be removed next, by means of a biting forceps. If one proceeds slowly and carefully and does not attempt to remove a large portion at one time, no trouble will be encountered. The bones that form the lateral limit of the ethmoid cells are very thin but are much denser than those that form the cells. They can be recognized readily by means of a probe, which should be used frequently in order to keep within the limits of the ethmoid cells at all times. Occasionally bone which is somewhat resistant is encountered in ethmoid cells. In this case one should work forward, beginning the removal of the ethmoid cells near the attachment of the middle turbinate. No force should be used, except in a downward and forward direction.

In cases in which one encounters a large fronto-ethmoid cell, the roof of which forms the floor of the frontal sinus, the partition usually offers very little resistance and is easily removed. If resistance is encountered, a probe or curet can nearly always be passed through the osteum and into the frontal sinus, and the obstructing partition can be broken with a down-

ward motion with the probe or curet. One now finds that there is a large direct opening into the frontal sinus. The floor of the sinus is formed by the superior internal wall of the orbit and there is nothing to obstruct free drainage into the nose. I have been able to pass a large dilator or the small end of a Faulkner ring curet directly into the frontal sinus. The last portion of the operation must be done without the aid of direct vision; consequently, caution should be used and no unnecessary force should be used at any time. If one pays close attention to the landmarks, studies the roentgenogram carefully, and keeps in mind the conformation of the sinus and that of the surrounding structures, no complications should be encountered.

In an early case, some irritation of the internal rectus resulted; this interfered with the rotation of the eye, and caused some diplopia. Apparently some inflammatory process developed from infection which penetrated through the orbital wall, or a hematoma formed. This irritation gradually subsided and there was a return to normal ocular function. After five years, this patient has had no recurrence of pain or discharge from the sinus.

In all cases in which an opportunity was afforded to observe the patients some months or years after operation, I have found a fairly large patent nasofrontal duct.

In cases in which complete ethmoidectomy and surgical treatment of the sphenoid sinus are indicated, it is a relatively simple procedure to extend the operation posteriorly by removing the remaining portion of the middle turbinate bone or by exenterating the ethmoid cells lateral to the turbinate bone, as indicated by the needs of the occasion. By keeping lateral to a line corresponding to the attachment of the middle turbinate bone, one will not injure the cribriform plate of the ethmoid bone, provided one proceeds with reasonable care and does not use any unnecessary force. The anterior wall of the sphenoid sinus can be removed rather easily and the floor of this sinus can be removed with the aid of a self-retaining nasal speculum.

Summary

In a large percentage of infections of the frontal sinus, good end-results can be obtained with the complete intranasal operation, and in most cases as much can be accomplished with this operation as with any other method. Great care must be taken to insure complete removal of all anterior ethmoid and fronto-ethmoid cells in cases in which disease has destroyed a portion of the anterior wall of the frontal sinus. In cases in which the bones that surround the nasofrontal duct have become thickened by disease or as the result of previous operation, the external route is the method of choice. If the results of surgical treatment of frontal sinusitis are judged by the appearance of the operative field or by the symptoms, less than one year after operation, a true conception of the efficiency of the surgical procedure will not be obtained.

Bibliography

1. Anderson, C. M.: External operation on the frontal sinus: causes of failure. Arch. Otolaryngol., 15:739-745, (May) 1932.
2. Jansen, A.: Zur Eröffnung der Nebenhöhlen der Nase bei chronischer Eiterung. Arch. f. Laryngol. u. Rhinol., 1:135-157, 1894.
3. Scheier: Quoted by Wright, Jonathan: A history of laryngology and rhinology. Ed. 2, Philadelphia, Lea and Febiger, 1914, p. 283.
4. Schaeffer: Quoted by Wright, Jonathan: A history of laryngology and rhinology. Ed. 2, Philadelphia, Lea and Febiger, 1914, p. 271.
5. Sewall, E. C.: External operation on the ethmo-sphenoid-frontal group of sinuses under local anesthesia. Arch. Otolaryngol., 4:377-411, (Nov.) 1926.
6. Winkler: Quoted by Wright, Jonathan: A history of laryngology and rhinology. Ed. 2, Philadelphia, Lea and Febiger, 1914, p. 283.

EDITORIAL

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BUSINESS MANAGER
J. R. BRUCE, Saint Paul

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Hay Fever in 1935

That the recent severe hay fever season was accompanied by an unusal number of unsatisfactory results from treatment is the common opinion of many of the doctors in the state. That this opinion is correct there is little question. The causes for the lack of good results are not easily found.

That this season was more severe than usual is unquestionable. A comparison of the pollen counts for the past five years, as carried on by A. O. Dahl of the Department of Botany at the University of Minnesota, shows that the concentration of ragweed pollen in Minneapolis was approximately 50 per cent heavier in 1935 than in any of the preceding years in which counts were made. The average maximum has been about fifteen hundred pollen grains per cubic yard of air. This year the maximum was twenty-two hundred. During the preceding four years the pollen concentration became high enough to cause symptoms on August 14, 15 and 16; this year the initial rise was on August 11. Usually the period when a large amount of pollen is present in the air lasts about three weeks, while this year this period was extended to four weeks. So the ragweed season began earlier, lasted longer and the pollen content of the air was as much as 50 per cent greater than usual.

Besides the factor of ragweed pollen itself, we are beginning to appreciate that fall hay fever is not a problem of ragweed pollen only, but that almost all hay fever sufferers are sensitive to other pollens, to specific dusts such as the animal danders, or to foods, and these may be the disturbing elements which prevent successful treatment.

To prevent the recurrence of such poor results as were obtained with too many patients this fall the following procedures seem to be indicated: more intensive treatment with ragweed pollen extract, especially during such a fertile summer as that just past; the addition of other pollens to which the patient is sensitive in inoculation; and the removal from the patient's environment, either physical or dietary, of other proteins which may cause the patient to have symptoms in spite of good ragweed desensitization.

H. M. SWEETSER, JR., M.D.

Part-Pay and Full-Pay Patients

Economic depression only serves to accentuate problems which are very real in the best of times. Free hospital care in tax supported institutions where the local profession donates its services has served to meet the problem of the care of the indigent in most localities. During hard times the load to hospital and staff is increased. To help lighten the load to the taxpayer some of the tax-supported hospitals in Minnesota have accepted part-pay patients who pay a nominal or very substantial part of the cost of their hospitalization, and the staff donates its services to this additional group. Such a patient is a pay patient as far as the hospital is concerned and charity as far as the profession is concerned. And the chances are that many of these part-pay hospital patients never know they are accepting charity from the doctors.

There should be no question as to the propriety of the attending physician's tendering a bill to a full-pay ward patient, even in a tax supported hospital. And yet we read that some of the larger hospitals of this type in Philadelphia have been in the habit of denying this right to the attending physicians in spite of a court action approving such procedure. To settle the controversy a joint committee, representing hospitals and profession, investigated the matter and decided to permit the attending physician to tender bills to full-pay ward patients up to a maximum of $25.00, with certain exceptions.

Of course the set-up in various localities differs. On the whole it does seem as though the attending physician should receive some compensation for his services to those who can pay something for hospital care.

"Throw-Away" Medical Magazines

The attention of the profession has been occasionally called, in the editorial columns of the *Journal of the American Medical Association*, to a type of magazine which is sent without charge to members of the medical profession, and it has been suggested that such magazines might well be thrown away—hence the term "throw-away magazine." These magazines are entirely commercial ventures and, while being commercial is not necessarily damning, there is usually a difference between a journal which is directed from a professional standpoint and one actuated from a commercial point of view. Such a journal depends entirely on advertising for its support, and its circulation, from the start, is what the owners wish to make it. Advertising rates are what the advertisers can be convinced the circulation justifies.

Some of these magazines contain only abstracts of medical articles which have appeared in medical journals—a duplication of the abstract departments of some of our national journals. Others contain articles emphasizing the economic side of practice and evidently are designed to undermine the policies of the organized profession.

One main objection to these parasitic magazines is that they afford an outlet for advertising proprietaries not acceptable to the Council on Pharmacy and Chemistry of the organized profession. This is decidedly against the best interests of medication in general.

If the throw-away magazines increase to any extent, it is conceivable that advertising in the regular medical magazines, which is essential to the publication of most medical journals, will suffer. It is felt in some quarters that even at the present time a deleterious effect is felt.

What is to be done about it? The most powerful weapon is public opinion in the profession. These magazines may either be thrown away or the publishers notified to discontinue mailing. The medical journals, too, may take united action, such as some have already done, by notifying these magazines to desist from abstracting their original articles.

While it is complimentary to MINNESOTA MEDICINE to have articles which have appeared in our columns republished in full or in abstract, credit being given the source of original publication, it is not so when it comes to the throw-away magazines. The original articles appearing in MINNESOTA MEDICINE are again to be copyrighted and the accompanying rights will be insisted upon in relation to throw-away medical magazines.

Rex Beach Writes Up Bonine

A recent issue of *Cosmopolitan* magazine carries an article by Mr. Rex Beach concerning the qualities of Dr. F. N. Bonine of Niles, Michigan, as a "famous benefactor of the blind" and a "Modern Miracle Man." In association with this article, *Cosmopolitan* magazine announces that "This article reflects the impressions of the author, and we publish it for its human interest value and not as carrying an endorsement." Dr. Bonine has apparently for years been doing a large business under the claim of dissolving cataract. Great numbers of people come to see him. They are charged $2 for the first visit and $1 for subsequent visits. The doctor practices in his shirt sleeves and the "miracles" are performed under circumstances which are calculated to impress by their simplicity. The usual method of treatment for patients with cataract is the prescription of eye drops which consist largely of boric acid and sodium salicylate in witch hazel water. There is not the slightest scientific basis for the claim that any cataract is benefited or removed by putting such drops in the eyes. Mr. Rex Beach and the *Cosmopolitan* are not rendering a service to mankind by their exploitation of so-called miracle men. They may cause a considerable number of sick people to spend money for transportation which might very well be spent in securing good medical care at the hands of competent physicians. Mr. Beach is himself hardly sufficiently trained in scientific medicine to have the slightest comprehension of the significance of testimonials or of the enthusiastic utterances of neighbors and friends in the villages which depend on the "miracle men" for such business as comes to them. (Jour. A. M. A., September 28, 1935, p. 1040.)

MEDICAL ECONOMICS

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association

B. J. Branton, M. D.
J. A. Moga, M. D.
W. F. Braasch, M. D., Chairman
J. C. Michael, M. D.
A. N. Collins, M. D.

What Can I Do For My County And State Medical Society?

Suggestion Number Two

1. Read the synopsis of Wisconsin's new plan for care of patients in the indigent and low income groups.

2. Take part in the discussion of this plan and the policy back of it at the next meeting of your county society.

Would contracts for care of the indigent be desirable in your county? Or credit agencies medically controlled to assist low income patients? Talk over the medical economic situation in your county. Formulate definite policies looking to definite programs. Ask your State Office for help. It may be time for you to act.

What have you done about Suggestion Number One, printed in these columns last month?

Have you interviewed the coach of your local high school debating team?

Have you offered him the assistance of the Minnesota State Medical Association for additional material for his debaters?

Have you secured literature on the subject yourself?

Remember that you are going to see that a representative of your society has an opportunity to give the medical viewpoint at the conclusion of the debates.

President Roosevelt has said, according to a recent issue of the *Journal of the American Medical Association*, that *"there will be no program for compulsory health insurance until public sentiment has crystallized in that direction. In the crystallization of public sentiment much will depend on this nation-wide series of debates, which will influence not only the minds of the adults of the next generation but those of hundreds of thousands of persons who will listen to the debates as they are publicly presented in innumerable villages, towns and cities."*

High School Debates

Preparations are going forward for the 1935 Inter-Scholastic Debates among approximately 102 schools in Minnesota.

The subject (as repeatedly stated in these columns):

RESOLVED: that the Several States should enact legislation providing for a system of complete medical service available to all citizens at public expense."

The list of schools that will enter this year is still incomplete but Mr. O. E. Smith, Anoka, director of the High School League, expects last year's 102 schools and perhaps a few others to enter.

At this time, therefore, it is particularly interesting to Minnesota physicians to note that high school debaters in Michigan will *not* debate on State Medicine.

Munitions in Michigan!

The subject selected by the National University Extension Association was proposed to Michigan educators just as it was to those in the 40 odd other states who participated last spring with this result:

Michigan high schools have for the subject of their interscholastic debate *"The Nationalization of Munitions!"*

It so happened that Prof. J. H. McBurney of the University of Michigan was one of the committee of five who phrased the proposition, too.

In Minnesota the subject had been accepted before the physicians were even aware that a meeting on this important question was to be held or that an opportunity might present itself to discuss the matter with local high school and college officials with a view to changing the subjects.

What About Minnesota?

Just what pressure was brought to bear in Michigan which led to dropping state medicine and substituting nationalization of munitions it would be interesting to Minnesota physicians to hear.

The fact that the Minnesota State Medical Association was not aware of these preliminary conferences at the University may well be taken as a serious reflection upon the organization's contacts with the high schools and colleges.

It emphasizes once again how important it is for our membership and the Auxiliary to keep in constant touch with all public activities.

If we had been more efficient in this respect we might have done as Michigan did.

The Doorbell Survey in Minnesota

The Health Survey, known popularly as the "doorbell census," will begin within a few months in Minnesota.

This is the survey of the amount and extent of chronic disease which is to be conducted under the auspices of the United States Public Health Service in 19 selected states this winter.

The survey will be financed under a special grant out of the funds of the WPA.

Assistant Surgeon General L. R. Thompson of the United States Public Health Service is directing the survey from its national headquarters at Detroit. His immediate assistants are Mr. B. St. John Parrot, formerly of the Milbank Fund, selected for an extensive experience with a variety of health surveys; also Mr. Clark Tibbets, who is likewise experienced in survey work having been employed on a number of government projects.

Bio-Statistician in Charge

The 19 selected states have already been divided into regions. The Great Lakes region of which Minnesota is a part, consists of Minnesota, Missouri, Illinois and Michigan. Its headquarters are in Chicago. Dr. A. W. Hedrich of Johns Hopkins University is regional director.

Dr. Hedrich, bio-statistician of Johns Hopkins University, has been loaned to the Public Health Service for the period of the survey which approximates six months.

As Minnesota newspapers and the *Journal of the American Medical Association* have already reported, five cities are to be selected for investigation in Minnesota. Four of them have already been determined. They are:

Minneapolis	Willmar
Saint Paul	Winona

A fifth will be announced later.

In each of the Twin Cities, 10,000 families will be enumerated. They will be so selected as to form a representative sample of the population. Smaller cities will be enumerated completely.

Minnesota Supervisor Selected

The enumerators who are to gather the information for the survey will be picked from the relief rolls. Only people who are well qualified for the task will be selected according to Dr. Hedrich, who spent some time in Minnesota making a preliminary investigation during October. Dr. Hedrich and the Health Service feel confident from experiences in other states that plenty of competent enumerators who will live up to their high standards for the work will be found on the relief rolls, individuals such as nurses, teachers, business women and the like.

The supervisor for Minnesota was just announced as this issue went to press. His name, according to press reports, is Kenneth E. Setre, of Minneapolis.

For each three enumerators there will be one office worker who will check and recheck the data submitted. These reports will then be sent to the Detroit headquarters where they will be edited and checked once more. After that, they will be ready for coding and interpretation by the statisticians.

The process of enumeration is expected to take about six months in Minnesota.

"Quality Not Quantity"

Quality not quantity is to be the watchword for all who have anything to do with the gathering of statistics according to Dr. Hedrich.

Every possible precaution will be taken to make the information accurate and of scientific value.

"Accurate information always leads to a constructive program," he observed in Minneapolis last week.

"It was not until the reasonably accurate statistics about the sickness and deaths among infants were provided by William Farr a century and a half ago in England that any constructive program was under-

taken to cut down the infant death rate in the British Isles.

"All of our progress in public health has waited upon accurate information.

"No Statistics on Chronic Disease"

"There are no accurate statistics as to the amount of chronic illness and its relation to employment and to medical facilities. Such statistics are necessary before we can make any great progress in attacking the control of chronic disease with all its ramifications.

"This information is tied up in the questionnaire with essential economic data hitherto not generally available; data on race, age and sex with reference to disease, on the mobility of the population and the health hazards of mobile population groups.

"There is no specific objective in this Survey other than the assembling of scientific information for the use of any agency that may have need of it.

"We hope that the medical profession will make use of the information gathered. The job of improving health is principally theirs. We hope especially that it will assist the physicians to establish themselves further in the periodic health examination field. We have every reason to believe that the facts gathered will make very evident the importance of this phase of preventive medicine."

Physicians to be Questioned

As already noted in a recent issue of the *Journal of the American Medical Association,* enumerators will be required to ask for the name of the family physician or the hospital or clinic and the physician, hospital or clinic will be asked to confirm the diagnosis given to the enumerator at the door.

This obviously involves the possibility that physicians will be asked to divulge confidential information without the consent of their patients.

That such procedure might involve the doctor in a legal difficulty has been discussed by physicians themselves and suggested as a real difficulty in the conduct of this survey as now planned.

It is Dr. Hedrich's belief that a satisfactory way out of this dilemma will be arranged. Possibly it will be a system of keying by which only the physician himself will know to what patients the numbers or symbols refer.

A statement relative to the medical coöperation to be invited will be issued shortly by the Surgeon General of the Public Health Service.

Medical Profession Will Watch

Whatever may be the official purpose of this survey, *medical observers are well aware that the statistics thus secured will be eagerly examined by the proponents of sickness insurance for any additional supportive aid and comfort they may provide.*

Assuredly the medical profession is interested in any scientific effort to secure further information about the extent of crippling illness in the United States. Medical men will offer any possible legitimate aid and coöperation to this end.

At the same time they will be on the alert to prevent the accumulation of inaccurate, biased statistics on the extent and quality of medical services, which will in their turn be used to fasten unnecessary and harmful reforms of medical practice by Congressional act upon the people.

On Privileged Information

The legal aspect of this matter of privileged information is of especial interest in view of the preliminary plans reviewed above for the "doorbell" survey.

Mason's Minnesota Statutes refer to it as follows:

Section 9814, Chapter 92, contains the provision that a *physician may not disclose in a court action or proceeding as a witness, without the consent of the patient, any information or any opinion based thereon which he acquired in a professional capacity and which was necessary to enable him to act in that capacity.* The statute further provides that "no oral or written waiver . . . of the privilege shall be binding except that the same be made upon the trial or examination where the evidence is offered or received."

Another reference is found in Section 5707, Chapter 35. This section authorizes the State Board of Medical Examiners to refuse to grant a license to, or revoke the license of, "any person guilty of immoral, dishonorable or unprofessional conduct." The words "immoral, dishonorable or unprofessional conduct" as used in this section are defined to mean, among other things, *"willfully betraying a professional secret."*

Statutes Show State Policy

Nothing in the above statutes is directly applicable to a situation which will arise out of the "doorbell" survey if the present plans are carried through in Minnesota.

They have been interpreted, however, as showing the spirit of Minnesota law on the subject of privileged information.

Dr. W. C. Woodward, director of the Bureau of Legal Medicine of the American Medical Association, is of that opinion as the following quotation from his letter on the subject will indicate.

"While the very comprehensive law in force in Minnesota that prohibits the disclosure of professional secrets in courts without the consent of the party affected is not directly applicable to the situation described in your letter (concerning the position of the physician in the 'doorbell' survey), it may be accepted, I believe, as evidence of the policy of the state with respect to professional secrecy."

Ethics and "Secrecy"

In "Principles of Medical Ethics" of the American Medical Association, the ethical position of the physician is clearly defined in Section 1 of the chapter on duties of physicians to their patients.

This section says:

"Patience and delicacy should characterize all the acts of a physician. The confidences concerning individual or domestic life entrusted by a patient to a physician and the defects of disposition or flaws of character observed in patients during medical attendance should be held as a trust and should never be revealed except when imperatively required by the laws of the state. There are occasions, however, when a physician must determine whether or not his duty to society requires him to take definite action to protect a healthy individual from becoming infected, because the physician has knowledge, obtained through the confidences intrusted to him as a physician, of communicable disease to which the healthy individual is about to be exposed. In such a case, the physician should act as he would desire another to act toward one of his family under like circumstances. Before he determines his course, the physician should know the civil law of his commonwealth concerning the privileged communications."

Counting Sick Men

(Chicago Tribune)

An interesting lay-slant on the Health Survey as it is to be conducted in Chicago is worth noting in the following editorial that appeared in a recent issue of the *Chicago Tribune:*

"The New Deal in Therapy"

"At a cost of $125,000 to the tax payers, a house to house canvas is to be made in Chicago to discover the relationship of illness and unemployment. A member of the medical faculty of Johns Hopkins has been sent here to run the show. When it is all over we shall know that some people are sick because they are unemployed and other people are unemployed because they are sick. A few of the shrewder heads in the community had suspected some such relationship but it will be nice to have the suspicions confirmed with neat tables of figures. . . .

"Naive laymen have been assuming for a long time that the art of medicine is the art of healing the sick. Obviously they were wrong. "Don't cure 'em; Count 'em,' is the New Deal's latest contribution to the relief of pain and disease among the unemployed. Apparently it is better to spend $125,000 to count a hundred thousand sick people than to cure 5,000 of them.

"Perhaps these remarks are unjust. Maybe after a sick man has been counted and carefully questioned as to his middle initial and when he first began to have his headaches, he will feel a lot better. Maybe."

Hospital Week-Ends

From the *Detroit Medical News,* August 12:

"A recent newspaper dispatch joyfully reports that as a result of adoption of the Group Hospitalization Plan, New Yorkers have established a new custom, the hospital week-end. Subscribers to this plan pay an annual fee amounting to three cents a day and are thereby entitled to three weeks' hospitalization without additional cost. Having paid, they quite naturally expect their money's worth. Dr. J. F. Kimball, originator of the plan, hastens to add, however, that these visits are not for the purpose of recreation but to avoid loss of time from business. We do not deny that this may be true. The fact remains, however, that illnesses which are amenable to elective week-end treatment are hardly to be considered of importance from the standpoint of public health. . . .

"The Group Hospitalization Plan already illustrates, in its comparatively brief existence, one of the glaring developments of sickness insurance, namely, increased sickness. There can be no doubt that malingering and 'compensation neuroses' play the major part in the increasing incidence of illness under insurance systems. Moreover, any plan which encourages hospitalization for petty complaints will, in the aggregate, swell, rather than decrease, the cost of medical care. And this, at the moment, is the real object of socio-economic planning in medicine.

"Another and by no means the least pernicious feature of hospital insurance schemes is that they provide the potential entering wedge for state medicine. It is generally recognized that these schemes tend to widen in their scope. . . .

"The *New York Post* puts the matter plainly—'Rather than obviating the need for national health insurance, the three cent plan paves the way for it.'

"Is group hospitalization the wolf in sheep's clothing?"

Concerning Birth Control

Activities of the American Birth Control League and of its constituent unit, the Minnesota Birth Control League, have had a tendency of late to cast some misunderstanding upon the action taken by the House of Delegates of the American Medical Association on the subject of contraceptives.

Neither the American Medical Association nor the Minnesota State Medical Association has ever taken any action or made any declaration for or against birth control.

The resolution passed by the House of Delegates of the American Medical Association at Atlantic City last June called for the appointment by the Board of Trustees of a committee which should make a study of the ultimate effect of the use of contraceptives upon health and general welfare of the people. No more, no less.

Following is the complete text as it was passed of the resolution with its preamble which should make this stand quite clear to members of the Birth Control League.

To Study Effects of Contraceptives

The House of Delegates declares nothing in the following resolutions to be interpreted as a declaration or action either for or against birth control:

WHEREAS, Under the stimulus of large non-medical groups, the general use of contraceptives is being advocated and encouraged despite the existing law, not only by the above mentioned groups but by commercial interests as well; and

WHEREAS, *The ultimate effect of these measures on the health and general welfare of the population of the United States is unknown if not questionable and should accurately and extensively be studied by the medical profession, in whose care the health of the people rests;* and

WHEREAS, The laws, both federal and local, governing the physicians in their advice to individual patients, where such advice is given as a therapeutic measure, seem to be complicated, not well understood and generally unsatisfactory and their interpretation difficult; therefore be it

RESOLVED, *That a special committee be appointed after due consideration by the Board of Trustees to study related problems and to present, at least a preliminary report to the House of Delegates of the American Medical Association at the 1936 Annual Session;* and be it further

RESOLVED, That the trustees be requested to appropriate the funds necessary to carry out the purposes of these resolutions.

The committee of the American Medical Association will meet shortly in Chicago and decide upon its program of study.

In the meantime medical men who are interested in the program and activities of the Minnesota league are being solicited to assist in League activities all over the state.

Four Clinics in Minnesota

Dr. Rae T. LaVake of Minneapolis was recently appointed state medical director for the Minnesota League according to News Letter No. 1, issued by the Minnesota Birth Control League September 10, this year.

It appears from this bulletin that Minnesota has only four Birth Control Clinics, two of them in Minneapolis, one in Saint Paul and one in Rochester. The number is very unsatisfactory to the heads of the League who compare Minnesota's four with Pennsylvania's twenty-five, California's thirty, and New York's thirty-five.

The Council Meets

Medical Defense

In accordance with a suggestion made by the Committee on Medical Economics and accepted by the House of Delegates in Minneapolis, a Medical Defense Committee was appointed at a special October meeting of the Council in Saint Paul.

Members are Dr. B. J. Branton, Willmar, member of the Committee on Medical Economics; Dr. W. L. Burnap, Fergus Falls, counsellor, eighth district, and Dr. W. H. Hengstler, Saint Paul, chairman of the Committee on Medico-Legal Affairs.

This committee will meet at stated times or at call of the chairman to discuss each malpractice case brought in this state against a member of the state association, to give advice to such member, if requested, and in general to assist in the conduct of his case.

Affiliate Membership

The new amendment to the Constitution of the state association, Article V, Section 4, which has to do with Affiliate Membership was changed as directed by the House of Delegates to make certain that nothing in the new ruling would

invalidate any affiliate memberships now in good standing.

The complete amendment reads as follows:

Affiliate members shall be those members of component medical societies:

1. Who, through disability, are unable to engage in the practice of medicine.

2. Who have retired from the practice of medicine.

Provided, however, that such member in either class, shall have first, upon his own request, been declared an affiliate member of such component society at its regular meeting, such action having been approved by the Council, and provided, further, that such Affiliate Membership shall automatically cease and revert to its previous status upon the termination of the disability or upon resumption of practice.

Nothing in this section shall in any manner invalidate an Affiliate Membership in good standing at the time of the adoption of this amendment.

Memorial

A memorial to the late Dr. Herman M. Johnson, to be placed in the Administration building at the University of Minnesota, was discussed by the Council. A committee was appointed to inquire into the possibilities for such a memorial and report to the Council at its next meeting.

What the Editors Say

Editorial comment occasioned by medical conventions is nearly always sympathetic and appreciative.

The following editorial comments appeared recently in the *Detroit News* and the *Detroit Free Press* respectively on the occasion of the meeting there. They are reprinted below in part for the interesting and significant points of view they reflect upon the part of the lay press.

Compliment to Doctors

Said the *News:*

". . . It is a compliment to the medical profession that we are amazed and indignant when doctors oppose social trends that would jeopardize their economic status. We have come to regard doctors as being primarily concerned with helping others."

For the Family Physician

From the *Free Press:*

"Naturally many things of consequence are being said at the convention sessions. But from the standpoint of general public interest nothing is likely to be more arresting than one of the remarks made by Dr. Charles H. Mayo at the opening meeting.

"Dr. Mayo prophesied the return of the family physician to his rightful place. 'The specialist will continue to have a place in society,' said Dr. Mayo, 'but the general practitioner who has been eclipsed by the specialist in the last decade is again coming to his own.'

Good News

"This is welcome news.

"We do not say so by way of minimizing the work or importance of the specialist. He is necessary and invaluable. He performs a service nobody else could give.

"But the overshadowing position the specialist has been occupying of late to the eclipse and discouragement of practitioners has not been good.

"The general practitioner also does a work, a most important one, that nobody else can perform.

"The competent family physician has a knowledge few specialists ever have a chance to acquire except in special instances.

"He knows his patients, their antecedents, background and mental and physical peculiarities. Often he knows their life history from babyhood. He can anticipate their reactions and their weaknesses and their immunities. He is their friend and father confessor.

"Study of a mere case history collected by a secretary is all a specialist often has in lieu of this; and it is a poor substitute.

"It would be a sad blow if specialists should disappear; and it would be an irreparable disaster if general practitioners were to die out.

"Both are needed but the second is fundamental."

New York's "Gradient Plan"

Notable among the many economic studies and experiments that are going on amoung county medical societies is the "Gradient Plan" proposed by the Medical Society of the State of New York.

For the purposes of this plan which is carefully worked out by a series of graphs people are divided into three groups: the "self-reliant"; the "partially self-reliant"; the "destitute."

A definite relationship is worked out between all welfare and health agencies and the medical profession for the care of each of these groups.

For the purposes of the diagram, definite lower limits of income are set to mark the "self-reliant" from the "partially self-reliant," and the latter from the destitute. The income figures, are, of course, based upon New York standards and are probably higher, therefore, than the same limits might be in the Middle West.

For Medical Banking

For the "self-reliant" and "partially self-reliant" groups who want to spread the cost of a major expense for health over a period of time, the New York committee suggests a new type of banking.

"The physician should be taken out of the banking business," says the committee. "He understands little or nothing about the functions of 'credit'; he has failed to use it for the betterment of either himself or the public."

On the other hand, bankers should be able to see readily the field of opportunity and service open to them in the provision of financial assistance for those who wish to spread the expense of major health and medical care over a period of income-time. With reasonable security and profit, millions of dollars could be put to work banking medical credits.

". . . The Federal Housing Act loans for house repairs, etc., have been extended to such needs as oil burners and ice boxes. Why should not the banking industry be given a like guarantee by our State or Federal government for financing health and medical credit needs?"

Minnesota State Board of Medical Examiners

"Rupture Expert" Warned Against Operating in Minnesota

Re: Mr. H. M. Shevnan, 6538 Talman Ave., Chicago, Ill.

The Minnesota State Board of Medical Examiners has issued a warning to Mr. H. M. Shevnan who advertises himself as a rupture shield expert, to refrain from operating in the State of Minnesota. Mr. Shevnan is not registered under the basic science law and neither is he licensed to practice medicine in the State of Minnesota. Mr. Shevnan had advertised in a number of southern Minnesota newspapers that he would be at the Saulpaugh Hotel at Mankato on September 24 and 25. The matter was referred to Mr. Brist who called upon Mr. Shevnan at Mankato. The law was explained to Mr. Shevnan and he stated that he would refrain from continuing his activities in this state, rather than have the matter taken into court. Mr. Shevnan stated that he originally planned to make stops at Albert Lea, Faribault, Winona and St. Cloud. He stated that he would cancel these arrangements and return to Chicago.

The State Board of Medical Examiners respectfully requests that it be notified at 524 Lowry Medical Arts Bldg., St. Paul, in the event any advertisements appear anywhere in the State of Minnesota indicating that Mr. Shevnan has returned or will return to this state.

Butterfield "Bone-Setter" Pleads Guilty at Fairmont

Re: State of Minnesota *vs.* Heppner

Peter D. Heppner, fifty-one years of age, who for a great number of years has resided on a farm two miles from Butterfield, Minnesota, entered a plea of guilty to an information charging him with practicing healing without a basic science certificate, on October 15, 1935, before the Honorable Julius E. Haycraft, Judge of the District Court at Fairmont, Minn. The complaint against Heppner was filed by Mr. Brist on behalf of the Minnesota State Board of Medical Examiners.

Heppner, since 1922, has represented himself as a "bone-setter." He has no medical education whatsoever and has been engaged in farming for the past thirty years. In 1934, following an investigation conducted by the Minnesota State Board of Medical Examiners, Heppner was warned to refrain from practicing healing in any manner in the State of Minnesota. Subsequently, he attempted to secure a license from the Medical Board to practice massage. He was unable to qualify and was denied the right to take the examination. The investigation disclosed that Heppner was operating on Monday and Tuesday of each week at the Edgewater Hotel at Fairmont. In his room were found numerous bottles, ointments, a cupping and bleeding apparatus and a device for giving electrical treatments. Heppner's patients paid him $1.00 for each treatment by leaving the money on the dresser in his room. In this manner, Heppner thought that he was immune from prosecution. After the facts had been presented to the Court, Judge Haycraft sentenced Heppner to a term of three months in the Martin County jail, ordered him to refrain from practicing healing in any manner in the State of Minnesota, and placed him on probation with instructions to report to the Court at the opening day of the March term. Judge Haycraft admonished Heppner to comply with the laws of this state in every respect, and told the defendant that unless he did so he would find himself in the county jail on very short notice.

Mr. C. L. Erickson, who is serving his first term as county attorney of Martin County, gave every assistance to the Board in the prosecution of this case.

Check Forgers Posing as Physicians Sentenced to Prison

Re: Edward Coats, alias Dr. Edward Coats, alias Edward W. DePuew, alias Edward W. DeCoatis, alias Edward LaRue, alias George Hall.
Re: Harry T. Workman, alias, Dr. L. E. DuBois.

Edward Coats — Harry T. Workman

The State Board of Medical Examiners wishes to advise the medical profession that two check forgers, who operated in St. Paul last spring and posed as doctors, have been apprehended and sentenced to prison. The check forger who posed as "Dr. Edward Coats" was sentenced on October 3, 1935, at Wichita, Kansas, to three years in the Federal penitentiary for forgery. At the time of his arrest, Coats was on parole from the Michigan state prison at Jackson, Michigan, where he had served time for forgery. He also had narcotics in his possession at the time of his arrest by the Wichita police. Coats is wanted at Newton, Kansas, for passing checks and also in St. Paul.

The fake doctor who passed five forged checks in St. Paul in the month of April, 1935, under the name

of "Dr. L. E. DuBois," was arrested in Cleveland, Ohio, and sentenced, on July 27, 1935, to an indeterminate term in the Ohio state prison, at Columbus. This faker passed a check at the Alverdes restaurant, the Florsheim shoe store and at three other places while in St. Paul.

At the time these men were operating in St. Paul, inquiry was made to the Medical Board by several persons, who had cashed these checks, as to whether these men were licensed to practice medicine in this state. Upon being advised that they were not licensed, they got in touch with the St. Paul police department. The Medical Board wishes to express its appreciation for the fine work done by Detective Lieutenant Frank J. Mondike, and his assistant, George I. Heim, of the St. Paul police, Check and Forgery Division. These men assisted in every way in locating these forgers and we quote Detective Lieutenant Mondike:

> "We intend to be bad medicine for fake doctors who operate in this city with forged checks."

The Medical Board solicits the coöperation of every member of the medical profession in immediately notifying the Board if any individuals representing themselves as doctors are passing worthless checks in their community.

Osage Iowa Quack Pleads Guilty at Albert Lea

Re: State of Minnesota *vs.* Henry H. Koller, alias "Prince Rudolph," alias "Dr." Koller.

Henry H. Koller, sixty-five years of age, formerly of Osage, Iowa, entered a plea of guilty, on October 10, 1935, to a charge of practicing healing without a basic science certificate before the Honorable Norman E. Peterson, Judge of the District Court at Albert Lea.

The State Board of Medical Examiners ordered an investigation of this man's activities following a report that Koller was practicing at the St. Charles Hotel, in Albert Lea. The investigation disclosed that Koller had been in Albert Lea, about a month prior to his arrest on October 5, and that he was representing himself to the people as Dr. Koller, specializing in eye, ear, nose and throat. Koller, claiming to be a descendant of Austrian nobility and the right to be addressed as Prince Rudolph, is neither registered under the basic science law of Minnesota, nor is he licensed to practice medicine. The investigation disclosed that Koller has the following record:

February 24, 1925, Koller was fined $100.00 and costs of $6.20 at Osage, Iowa, on a charge of practicing medicine without a license. Koller was giving electrical treatments and furnishing pills and had charged a patient $140.00 for two weeks treatment.

May 12, 1925, Koller pleaded guilty, at Osage, Iowa, to a charge of rape committed on a seventeen-year-old girl from Mower County, Minnesota, and was sentenced to a term of not to exceed ten years at the Iowa state prison at Fort Madison. April 23, 1930, he was released from prison.

February 26, 1931, Koller pleaded guilty to a charge of "resorting to a hotel for the purpose of prostitution and lewdness." Koller had left the state of Minnesota with the wife of a man from Martin County, and was apprehended at McIntire, Iowa, and sentenced to serve not to exceed five years in the Iowa state prison at Fort Madison. March 16, 1934, he was released from prison.

March 5, 1935, Koller was permanently enjoined from practicing medicine in the state of Iowa.

March 6, 1935, Koller's license as an optometrist in the state of Iowa was revoked.

Upon being questioned, Koller admitted having been declared insane, but that the finding was reversed by the District Court of Iowa. Following the presentation of all these facts to the Court, Judge Peterson imposed a sentence of six months in the county jail of Freeborn County. Judge Peterson told the defendant that he was no credit to the community and ordered him escorted to the Iowa state line. This was done because Koller is not a resident of the state of Minnesota, and had been a public charge of Mitchell County, Iowa, for four months previous to coming to Albert Lea. The Court forbade the defendant to practice healing in any manner in the state of Minnesota, and further informed him that if he returned to this state he would have to serve this jail sentence. Koller was immediately returned to the authorities at Osage, Iowa.

The State Board of Medical Examiners wishes to express its appreciation for the prompt and efficient coöperation shown in this case by Chief Miles Norton of the Albert Lea police department, Sheriff Helmer C. Myre of Freeborn County, and County Attorney Elmer R. Peterson.

Pyrethrum Ointment

An ointment containing an extract from powdered pyrethrum flowers (Chrysanthemum cinerariæfolium). The extract is obtained by treating powdered pyrethrum flowers with a hydrocarbon oil of the kerosene type; this extract is then incorporated into an ointment base composed of hydrous wool fat, petrolatum and paraffin. The finished product contains 27 per cent of the active extract and 73 per cent of ointment base. This product has been shown to be an effective agent in the treatment of scabies, and except in rare instances it does not produce dermatitis with resultant exfoliation. Upsher Smith Company, Minneapolis.

Can the Leopard Change Its Spots?

The "patent medicine" interests have decided that another housecleaning is called for. In August, 1934, there was published in the *Editor and Publisher* an article entitled "Proprietary Association Begins Clean-Up." In this it was stated that the Proprietary Association—the present name for the organized "patent medicine" interests—had created an Advisory Committee on Advertising, which would draw up an "Outline of Ethical Practices in Proprietary Advertising." In June of this year the New York *Times* reported that the Proprietary Association was about to "launch a comprehensive program of research." The same article stated that Dr. Frederick J. Cullen (formerly Chief of Drug Control of the Department of Agriculture), general representative of the Proprietary Association, was also serving, ex officio, as secretary. There has recently been issued (August 28, 1935) what is presumably one of the first pieces of work in this field. In April, 1933, the Department of Agriculture issued under Dr. Cullen's name a bulletin warning the public against the coated laxatives that contain phenolphthalein. Now the Proprietary Association, under Dr. Cullen's name, sends Bulletin 6365 to all its members, in which Dr. Cullen states that the government bulletin (April, 1933) issued by him was based on information gathered from medical textbooks and from a report made by a physician in one of the medical journals (the report of the case of a small child who ate a complete box of the "candy laxative" known as "Ex-Lax" and promptly died). It appears that Dr. Cullen today has decided, "a more exhaustive study of the effects of phenolphthalein since the issuance of that article has resulted in a change in my opinion as to its harmful effects." Undoubtedly the "patent medicine" interests are on the defensive and presumably will spare no expense to convince a gullible public that its business is a public-spirited activity. But those who have followed critically the devious methods of the "patent medicine" business for more than a quarter of a century may be excused for holding to the well established opinion that the leopard does not change its spots. (Jour. A. M. A., September 21, 1935, p. 971.)

OF GENERAL INTEREST

Dr. C. E. Johnson, formerly of Cambridge, has recently located at Pine River.

* * *

Dr. M. Hansen of Ada has recovered from a severe case of erysipelas contracted in August.

* * *

Dr. Herbert Davis, Saint Paul, is rapidly recovering from a gallbladder operation performed at Rochester.

* * *

Dr. L. Gordon Samuelson of Mankato recently bought the beautiful home of Clifford Babcock on Oak Knoll.

* * *

Dr. A. J. Wentworth, who has been absent from the Mankato Clinic for one year, will return the first of November.

* * *

Dr. George Bohl of Ada was a member of the group of Great Northern surgeons that met at Glacier National Park in July.

* * *

Dr. Thomas H. Dickson was recently appointed Medical Director of the Minnesota Mutual Life Insurance Company to succeed Dr. C. N. McCloud.

* * *

Dr. Theodore Loken of Ada, a recent benedict, attended a course of instruction in September at the Cook County Graduate School of Medicine in Chicago.

* * *

Dr. E. D. Greenberger, who has been taking Dr. A. J. Wentworth's place in the Mankato Clinic, will go to McAlister, Oklahoma, to take over an x-ray practice.

* * *

Dr. and Mrs. A. J. Button of Pine River returned recently from a trip to the Pacific Coast, visiting en route Glacier and Yellowstone Parks, Seattle and Portland.

* * *

Dr. James T. Priestley of the Mayo Clinic gave an address Monday, October 28, before the Blue Earth County Medical Society. His subject was "The Surgical Treatment of Peritonitis."

* * *

Dr. Kenneth E. Bray, son of Dr. C. W. Bray of Biwabik, and a graduate of the University of Minnesota, 1934, has joined Dr. A. T. Laird's staff at the Nopeming Tuberculosis Sanatorium.

* * *

Dr. W. S. Middleton of Madison, Wisconsin, Dean of the Medical School, was the guest speaker before the Saint Luke's Hospital staff, Duluth, at its regular September meeting. He discussed oxygen therapy.

* * *

Dr. Charles B. Wright, Minneapolis, has been appointed a member of the Editing and Publishing Committee of the Minnesota State Medical Association to fill the vacancy due to the illness of Dr. A. S. Hamilton.

* * *

Dr. Adolph M. Hanson, Faribault, Minnesota, addressed the Minneapolis Clinical Club, October 10, on "Some Aspects of the Thymus and Pineal Problems." Honorary membership in the Minneapolis Clinical Club was conferred upon Dr. Hanson at this meeting.

* * *

Superintendents Sister M. Patricia of Saint Mary's Hospital and James McNee of Saint Luke's, Duluth, attended the sessions of Hospital Administrators and the American Hospital Association, late in September, at Saint Louis.

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Correction.—In the article entitled "Pathologic Conditions in the Newborn" by Thomas Mayers, M.D., which appeared in the October, 1935, number of MINNESOTA MEDICINE, the illustrations for Figures 1 and 2 (page 659) are reversed. The legend for Figure 1 as it appeared in the journal applies to Figure 2, and vice versa.

* * *

Much progress has been made in Norman County in the immunization of school children against smallpox and diphtheria. Vaccination clinics were held May 18 in Twin Valley, Ada and Halstad when more than 2,200 children were vaccinated. The state furnished the toxoid, county organizations supplied funds for the vaccine, and the doctors did the vaccinating for nominal fees.

* * *

Norman County has handled its own relief cases for the past two months. The local medical group has had cheerful coöperation from the relief authorities. The doctors have always done their best to keep their pations on the "pay their own bills" schedule whenever possible, knowing full well that those who pay something, however small, are better patients than those who pay nothing.

* * *

Dr. Fred G. Carter, superintendent of Ancker Hospital, Saint Paul, since December, 1924, will leave November 15 to accept the position of superintendent at Christ Hospital in Cincinnati. It is understood that Dr. S. R. Lee, formerly an assistant to Dr. Carter, but at present superintendent of the state asylum at Willmar, will succeed Dr. Carter.

* * *

The Northwestern Clinic at Crookston celebrated its fifteenth anniversary October 10, 1935. A banquet was served at the Hotel Crookston for the staff, employees, and wives of the physicians. The Clinic was opened in 1920 by Drs. M. O. Oppegaard and O. E. Locken, together with Dr. C. D. Mitchell, dentist. Members added to the staff since that, and now associated in practice, are Drs. C. L. Oppegaard, L. L. Brown, and W. F. Mercil. The Clinic employs a total of fourteen at present.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

Medical Broadcast for November

The Minnesota State Medical Association Morning Health Service.

The Minnesota State Medical Association broadcasts weekly at 11:15 A. M. every Tuesday over Station WCCO, Minneapolis and St. Paul (810 kilocycles or 370.2 meters).

Speaker: William A. O'Brien, M.D., Associate Professor of Pathology and Preventive Medicine, Medical School, University of Minnesota.

The program for the month will be as follows:

November 5—Socializing Children.
November 12—Heart Murmurs.
November 19—Digestive Complaints.
November 26—Functions of the Teeth.

State Association Meeting

The Committee on Scientific Assembly for the 1936 annual meeting of the Minnesota State Medical Association held its first meeting of the year at the Nicollet hotel in Minneapolis, Saturday, October 26.

Members of this committee are: Dr. W. W. Will, Bertha, president elect of the state society, chairman: Dr. Gordon B. New, Rochester and Dr. T. H. Sweetser, Minneapolis, chairman and secretary, respectively, of the Surgical Section; Dr. F. J. Hirschboeck, Duluth, and Dr. C. N. Hensel, St. Paul, chairman and secretary, respectively, of the Medical Section; Dr. W. A. O'Brien, Minneapolis, chairman of Clinical Demonstrations; Dr. E. G. Bannick, Rochester, chairman and Drs. E. H. Rynearson, D. L. Wilbur, H. K. Gray and J. T. Priestley, members of the Local Arrangements Committee, and Dr. E. A. Meyerding, St. Paul, secretary of the association.

The 1936 meeting will be held at Rochester, May 3, 4, 5 and 6.

Following the Saturday night meeting, members of the above committee issued an invitation to all members of the Association who wish to present papers at the annual meeting to send their names at once to the Secretary's office, 11 W. Summit Ave., St. Paul. The committee will hold its next meeting early in December.

Sixth Councilor District Meeting

Several hundred medical men from the sixth councilor district, including members of the Wright County, Stearns-Benton, Red River Valley, Clay-Becker, Kandiyohi-Swift-Meeker and East Central Minnesota Medical Societies together with all officers and committee chairmen of the state association will attend an important councilor district meeting to be held at St. Cloud, November 21.

Dr. Morris Fishbein, Chicago, editor of the *Journal of the American Medical Association,* will be the principal speaker at this meeting which is to be held in honor of President-elect W. W. Will, Bertha, of the Minnesota State Medical Association and Dr. E. J. Simons, Ah Gwah Ching, newly elected councilor of the district.

There will be a dinner for the physicians at the St. Cloud hospital to be followed by a meeting at the Assembly rooms of the Technical High School. Members of the Women's Auxiliary, dentists, druggists and her interested persons will be invited to attend this latter meeting. On the program, beside Dr. Fishbein, are Dr. C. B. Wright, of Minneapolis, trustee of the American Medical Association, and Dr. J. A. Myers, Minneapolis, Professor of Preventive Medicine at the University of Minnesota and Dr. S. A. Slater, Worthington, director of Southwestern Sanitarium.

On the same evening Dr. Fishbein and Dr. Myers will also address a public health meeting to be held jointly by the Stearns County Public Health Association and the Minnesota Public Health Association in the Auditorium of Technical High School.

Rice County Society

Dr. Leo G. Rigler of the University of Minnesota addressed the Rice County Medical Society at a meeting held in the Faribault Clinic rooms at Faribault, Wednesday, October 9, at 7:45 p. m.

Dr. Rigler's subject was "The Development of the Use of Opaque Medica in Roentgen Diagnosis."

The program also included reports by Dr. F. J. Lexa and Dr. S. B. Haessly on the last state session and on the bureau of collections, respectively.

Upper Mississippi Society

The Upper Mississippi Medical Society held their regular meeting October 17, 1935, at the Commercial Hotel, Wadena.

Following a 6 o'clock dinner the members were addressed by Dr. W. W. Will, President of the State Medical Association, and Dr. H. S. Diehl, Dean of Medical Sciences, University of Minnesota. Dr. C. B. Wright, Minneapolis, read a paper entitled "Abdominal Symptoms of Heart Disease," and Dr. Theodore Sweetser, Minneapolis, spoke on "Hydronephrosis, Diagnosis and Treatment."

WOMAN'S AUXILIARY

Mrs. F. J. Elias, *President*, Duluth, Minn.
Mrs. L. W. Barry, *Editor, Press and Publicity*, 2193 Sargent Ave., St. Paul, Minn.

The first meeting of the new board of the Women's Auxiliary to the Minnesota State Medical Association was held Tuesday, October 8, 1935, at the home of Mrs. F. J. Elias, State President, at Duluth, Minnesota, twenty-four members being present. The budget was discussed and approved, and appointments of committee chairmen were ratified.

Dr. W. A. Coventry, president of the Minnesota State Medical Association, reviewed the social problems of the day as they affect the practice of medicine.

Mrs. Florence Fitzgerald, from the office of the State Medical Association, spoke on the 1935 High School Debate, the subject of which is:

Resolved: That the several states should enact legislation providing for a system of complete medical service available to all citizens at public expense.

The Board of the Auxiliary voted to coöperate with the doctors of the State of Minnesota in disseminating information on this subject, and to adopt it as one of the major projects of the year.

The Board also voted to coöperate with the Minnesota Public Health Association in sponsoring the annual high school radio contest held in connection with the 1935 Christmas Seal educational campaign. The subject for this year's talk is "Fight Tuberculosis with Modern Methods." Every Junior and Senior high school in Minnesota is eligible to enter one student's manuscript which has previously been delivered in public by the author. The contest will close November 23, and daily broadcasts over WCCO of the ten winning talks will begin about December 10. A committee of doctors and public speaking experts will serve as judges.

The State Auxiliary will again present the awards for the contest, including engraved gold medals to each student chosen to broadcast his talk, and a specially designed radio plaque to the school whose student wins first place in the broadcasting. Several counties have presented an award to the winner of the contest in their county, and plan to do so again. Ramsey County donated a loving cup to the winner last year, and Hennepin County offered the choice of a watch or two weeks at camp as its award to the winner in Hennepin County.

For the first time this year special prizes are being offered the schools which put on the best educational campaign on tuberculosis in the local preliminary contest. These include equipment to be awarded to the manual training department of the two schools judged best.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

The Pathology of Internal Diseases. Second Edition. William Boyd, M.D., M.R.C.P., Ed., F.R.C.P., etc., Professor of Pathology, University of Manitoba, Pathologist to Winnipeg General Hospital, etc. 904 pages. Illus. Price, cloth, $10.00. Philadelphia: Lea & Febiger, 1935.

Diseases of the Nose and Throat. Charles J. Imperatori, M.D., F.A.C.S., Professor of Clinical Otolaryngology, New York Post-Graduate Medical School, Columbia University, and Herman J. Burman, M.D., Instructor of Clinical Otolaryngology, New York Post-Graduate Medical School. 723 pages. Illus. Price, cloth, $7.00. Philadelphia: J. B. Lippincott Company, 1935.

APPARATUS ACCEPTED. By the Council on Physical Therapy of the American Medical Association, 1935.

Minnesota Medicine has received from the American Medical Association a small pamphlet containing a list of the apparatus accepted by the Council on Physical Therapy, the first one published under the direction and supervision of the Council. In addition to the list and description of accepted apparatus, the pamphlet contains indications for the use of each type and a statement relative to efficacies and dangers.

This pamphlet is a real contribution on the part of the American Medical Association in behalf of rational therapeutics—an effort to help place physical therapy on a sound, scientific basis for the benefit of the medical profession.

One of the purposes of the Council on Physical Therapy is to protect the medical profession, and thereby the public, against inefficient and possibly dangerous apparatus and against misleading and deceptive advertising in connection with the manufacture and sale of devices for physical therapy.

Apparatus Accepted includes all the devices accepted by the Council prior to May, 1935. Any physician can obtain this pamphlet free by writing to the Secretary, Council on Physical Therapy, A. M. A., 535 North Dearborn Street, Chicago, Illinois.

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